Original Article

Renal Changes in Burn Related Fatalities- An Autopsy based Prospective Histopathological Study from Tertiary Care Centre

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Abstract

Background: The intricate pathophysiological response along with burn induced kidney damage in Burn Injury needs to be better understood with a pathological study of the patient's organs, which not only can shed light on disease progression and associated morbidity & mortality but also guide us in treatment response.
Methods: A unicentric, prospective cross-sectional study was carried out in the Department of Pathology in collaboration with department of Forensics & State Medicine, Burdwan. Autopsy samples of both kidneys of Burn Injury (n=42) were processed, stained with H & E and reported by two Pathologist. Statistical evaluation was done in percentage, mean±standard deviation, Chi-square test and Fisher's exact test.
Results: Maximum number of study population belonged to 21-40 years age group (76.19%). Most of them were females (85.71%), married (71.43%), Hindu by religion (73.81%) and belonged to rural population (80.95%). On Microscopic examination, 80.95% of study population had severe tubular necrosis amongst which only 45.24% survived for <72 hours. Severity of tubular necrosis is negatively correlated with duration of survival which was statistically significant. Desquamation of tubular epithelium and tubular cast formation (n=14, 33.33%) showed statistically significant correlations with increasing burn severity (>90%TBSA) and negative correlation with duration of survival. Chronic interstitial inflammation (n=19, 45.23%) was found to be positively correlated with duration of survival with P-value of 0.0001.
Conclusion: Burn-induced kidney damage restricts renal blood supply, causing epithelial degeneration and necrosis, with tubular cast formation and desquamation of tubular epithelium being significant indicators of injury severity and potential early mortality. Comprehending the complex interplay between burn severity, survival time, and kidney changes at different levels is vital, necessitating further investigation into the relationship between burn extent, survival duration, and subsequent renal alterations.

Keywords: Burn Injury; Renal Autopsy; Tubular necrosis; epithelial desquamation; tubular cast

Introduction

Burn is the second largest injury after road traffic accident in India. Almost 10% case of burn injury is life threatening requires hospitalization and almost half of those hospitalized succumb to their injuries. The most common cause of death in burn injuries is sepsis. Other causes are further complications of sepsis such as inhalational injury, infection and shock. [1, 2]

The pathology of burn injury is multifactorial having a complex pattern, as the pathology goes beyond the skin (commonly quantified as a percentage of the total body surface area involved (%TBSA)) and reaches into various internal organs. Different pathological, biochemical, immunological and microbiological responses drive the case of burn and ultimately

cause systemic inflammatory response syndrome (SIRS). The result is manifested clinically as multiple organ dysfunction syndrome (MODS) and finally death. The most commonly involved organs in burn patients are lungs, liver, kidney and the upper gastro-intestinal tracts. [3]

The intricate pathophysiological response along with rapid involvement of various organ system associated with burn injury can be understood better with a pathological study of the patient's organs. [4] By studying the tissue and organs of deceased individuals, pathologists can gather how diseases progress, their impact on the body and the underlying factors that led to their death and why certain treatments may or may not have worked. [5] Not only that, Soyka P et al established a time table of danger phases in burn patients and concluded that the badly burned patient should not be overhydrated in the initial phase of therapy based on the findings of presence of organ edema resulting in death due to unknown cause. [6]

Burn injury is associated with a high risk of acute kidney injury (AKI) with a prevalence of AKI among patients with burns of 9–50%. Despite an improvement in burn injury survival in the past decade, AKI in patients with burns is associated with an extremely poor short-term and long-term prognosis, with a mortality of >80% among those with severe AKI. [7] It usually occurs immediately after burns and is mostly due to reduced cardiac output, which is mainly caused by fluid loss. This is usually caused by delayed or inadequate fluid resuscitation but may also result from substantial muscle breakdown or haemolysis and it is usually reversible. [8]

Acute tubular necrosis (ATN) is the most common cause of acute kidney injury, and renal outcomes closely correlate with its severity. ATN is usually diagnosed clinically, with biopsy reserved for cases where an alternative cause is suspected. The most frequent histological finding in ATN is tubular cell sloughing, followed in order by tubular epithelial flattening, tubular dilatation, cell necrosis, regenerative changes, vacuolization, and loss of the brush border. [9]

Burn-related acute kidney injury (AKI) is classified as early or late based on timing and cause. Early AKI occurs within 0–3 days after injury, usually during initial resuscitation, and is associated with hypovolemia, inflammation, tissue damage, altered protein release, cardiac dysfunction, and the severity of burn shock rather than fluid volume alone. Late AKI develops between 4–14 days post-burn and is commonly linked to sepsis, multi-organ failure, fluid overload, or nephrotoxic drugs. [10]

As mortality associated with kidney injury is very high, the present study was carried out to evaluate the histopathology of kidney in cases of burn Injury and also, to correlate the changes in histopathology of kidney with severity of burn injury and duration of survival post burn injury.

Material & Methods

This was a unicentric, prospective cross-sectional study where All burn victims admitted to the Emergency of Burdwan Medical College & Hospital or brought dead in the mortuary in Department of Forensic and State Medicine, BMCH, Burdwan, West Bengal from January, 2023 to December, 2023 were included after obtaining IEC approval. A total 42 cases of burn death autopsy interval of less than 12 hours were included by complete enumeration method for sample collection. A pre validated and predesigned proforma was used for recording details of victim's profile.

A medicolegal autopsy was done under command of either police as per section 174 CrPC or magistrate as per section 176 CrPC. In most cases, police officials or magistrate identified body before autopsy. Autopsy was done in all fatal burn cases irrespective of age, gender and mode of burn. Autopsy findings were recorded in standard formats [PM Report vide West Bengal form number 5372]. Cases of scalds, electrocution, mechanical injuries, auto-lysed tissue, decomposed body and improperly stained sections were excluded from the study. Study. All histopathology slides underwent routine quality control prior to evaluation. Sections were prepared using standardized tissue processing and staining protocols. Slides were initially screened for adequacy of tissue, uniform section thickness, and staining quality (hematoxylin–eosin contrast, nuclear and cytoplasmic clarity). Sections showing inadequate fixation, tissue folding, excessive artifacts, or suboptimal staining that could interfere with interpretation were classified as improperly stained and excluded from analysis.

Autopsy samples of both kidneys were sent to Department of Pathology in 10% formalin solution, where it was processed, stained and reported. Inter-observer variability was addressed by having two independent pathologists evaluate the specimens in a blinded manner, without access to each other's findings. Reporting discrepancies were resolved through joint review and consensus. Concordance between observers was assessed using Cohen's Kappa (κ) to ensure consistency and reliability of the interpretations.

Data was put into Microsoft Excel sheet and statistical evaluation was done. Results were represented in percentage, mean, standard deviation. Chi-square test and Fisher's exact test were done wherever required. Statistical significance was considered if $p<0.05$.

Results

The study population consisted of 42 autopsy cases from January to December 2023, all of which were determined to be deaths resulting from burns. A comprehensive gross and microscopic examination of both kidneys was performed in each of these cases.

Maximum number of study population belonged to 21-40 years age group (76.19%) followed by 1-20 years, 41-60 years (9.52%) and 61-80 (4.76%), with mean age of 30.43 $\pm$ 12.39 years. Most of them were females (85.71%), married (71.43%), Hindu by religion (73.81%) and belonged to rural population (80.95%).

The study revealed that over half of the burn victims (59.524%) had substantial burns affecting 60-90% total body surface area (TBSA). Notably, nearly half of the fatalities (47.61%) occurred between 1 and 7 days after the burn injury (Chart 1). The main causes of death were attributed to septicemic shock, hypovolemic shock, or various infections.

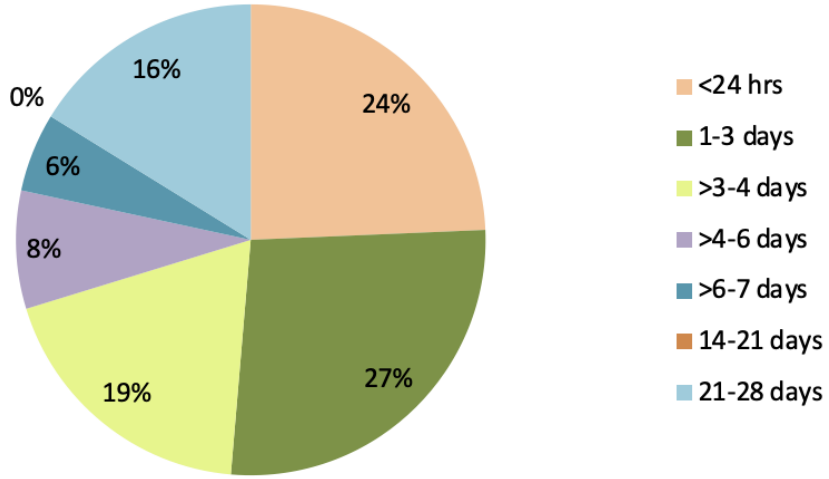

Figure 1: Duration of survival in hours and days after burn injury.

Kidney Changes-Gross Examination & Microscopic Examination in respect to %TBSA

The mean renal length of right and left kidney was found to be 103.67 mm and 104.11 mm respectively in <24 hours after burn injury and was 104.74mm and 105.68mm respectively after 24 hours. The average thickness of the right and left renal cortex was 9.23mm and 9.31mm respectively in <24 hours and was found to be 8.66mm and 9.22mm beyond 24 hours. Both the findings did not show a statistically significant relationship with the severity of burn injury or how long the burn victims survived. (Figure 1a and 1b)

Distribution of kidney findings Namely Surface congestion, Desquamation of Tubular Epithelium, Tubular Cast, Interstitial Congestion and Glomerular congestion (Figure 1c and 1d) were studied across different severities of burn injuries (Table 1) and calculated statistically using Pearson's correlation coefficient (Chart 2). All the mentioned changes show positive correlation with %TBSA. Desquamation of tubular epithelium and tubular cast formation show strong, statistically significant correlations with increasing burn severity (%TBSA). Other findings namely surface congestion, interstitial congestion and glomerular congestion do show some increasing trend, but the correlation is not statistically significant.

Kidney Changes- Microscopic Examination in respect to duration of survival

Certain kidney findings namely, cloudy degeneration, desquamation of tubular epithelium, tubular cast and interstitial edema was also studied in relation to different duration of survival categories in days (Table 2) and calculated statistically (Chart 3). All the mentioned changes show negative correlation with the duration of survival. Tubular Cast and Interstitial Edema show strongest correlations, with p-values close to 0.05 while Desquamation of tubular epithelium and Cloudy Degeneration moderate to strong and moderate negative correlation (not significant) respectively. Only presence of interstitial chronic inflammation was found to be positively correlated with duration of survival with P-value of 0.0001(Fisher-exact test) denoting strong statistical significance.

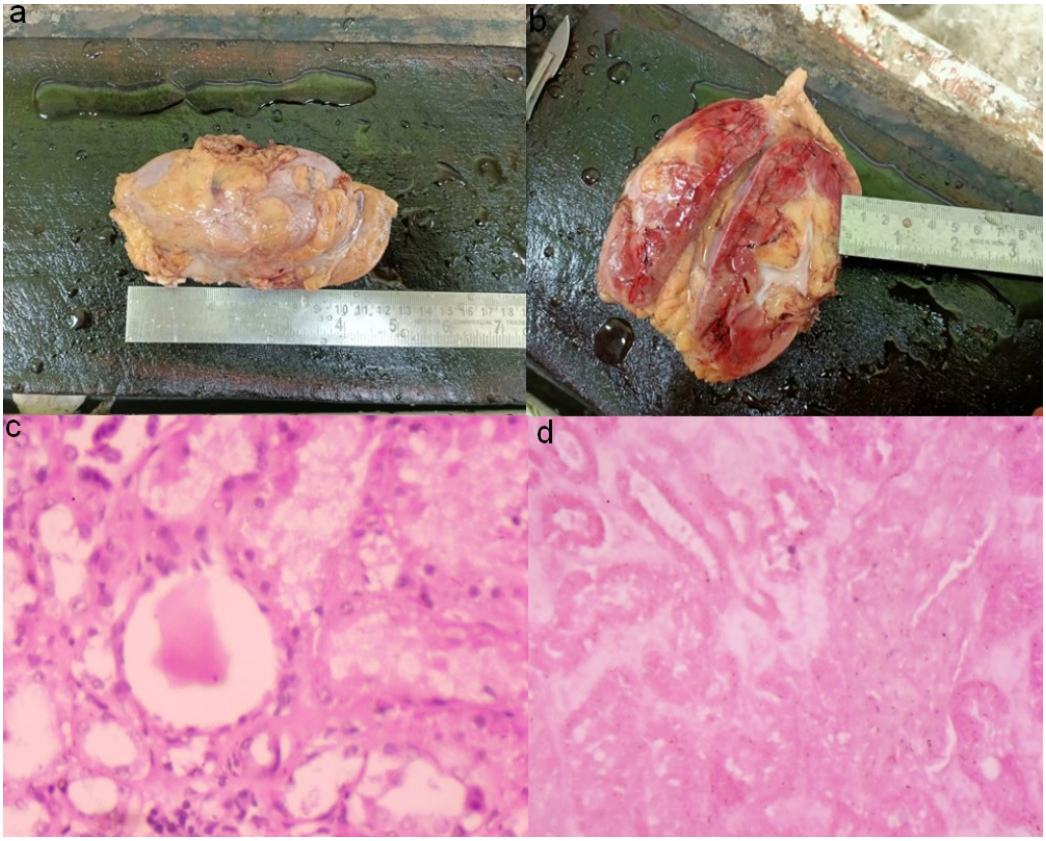

Figure 2: (a & b): Gross photograph of kidney showing the length and cortical thickness along with interstitial congestion. (c & d) Microscopic picture of kidney showing presence of tubular cast, desquamation of tubular epithelium and tubular necrosis.

Table 1: Distribution of kidney changes across percentage of TBSA burn among study population.

Kidney Changes		30-60% TBSA	>60-90% TBSA	>90% TBSA	Total n=42	Correlation (r)	P value
Surface Congestion	Present:	1	20	8	29	0.057	0.6895
	Absent:	2	5	6	13		
Desquamation of Tubular Epithelium	Present:	0	11	14	25	0.766	0.1297
	Absent:	3	14	0	17		
Tubular Cast	Present:	0	9	14	23	0.774	0.0309
	Absent:	3	16	0	19		
Interstitial Congestion	Present:	0	23	14	37	0.638	0.5140
	Absent:	3	2	0	5		
Glomerular Congestion	Present:	0	22	14	36	0.538	0.4941
	Absent:	3	3	0	6		

Table 2: Presence or absence of certain kidney findings in relation to different duration of survival categories (in days).

Kidney Changes		<24 hrs	1-3 days	>3-7 days	14-21 days	21-28 days	Total n=42	Correlation (r)	P value
Cloudy Degeneration	Present	3	8	8	2	3	24	-0.575	0.3106
	Absent	6	2	4	4	2	18		
Desquamation of tubular epithelium	Present	9	10	1	3	2	25	-0.660	0.2259
	Absent	0	0	11	3	3	17		
Tubular cast	Present	9	10	2	2	0	23	-0.822	0.0880
	Absent	0	0	10	4	5	19		
Interstitial edema	Present	5	6	4	2	3	20	-0.824	0.0864
	Absent	4	4	8	4	2	22		
Chronic Inflammation	Present	0	8	0	0	11	19	0.752	0.0001
	Absent	0	23	0	0	0	23		

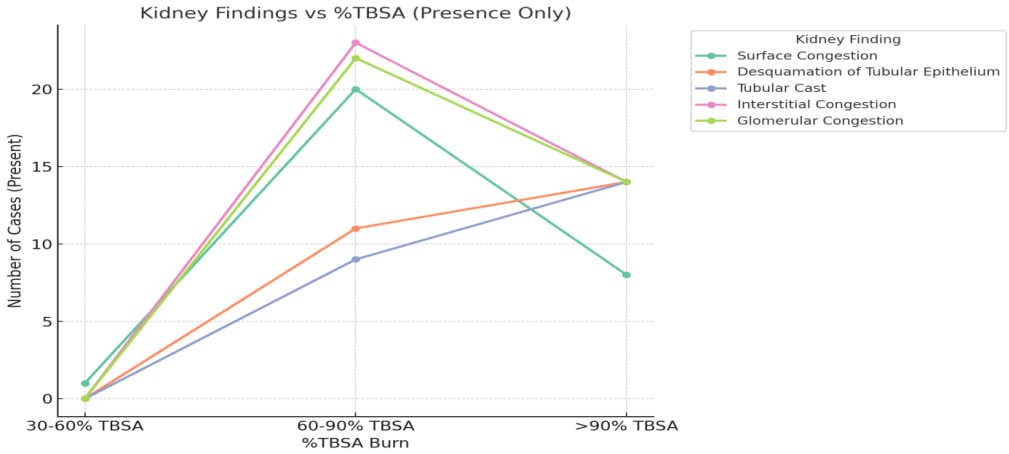

Figure 3: Chart showing how the presence of each kidney finding correlates with increasing %TBSA (burn severity) among study population.

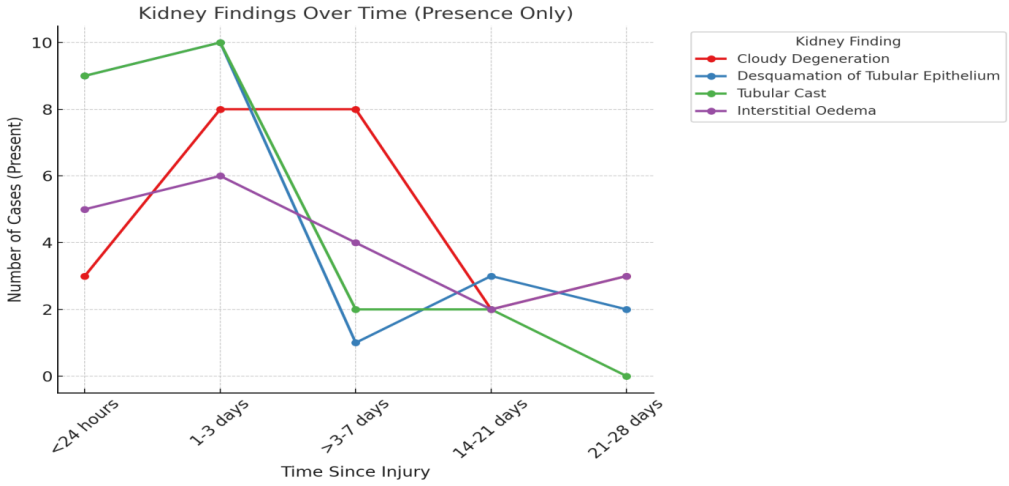

Figure 4: Trends observed between certain kidney findings and different duration of survival categories in days.

Degree of Tubular necrosis and its association with %TBSA and duration of survival

In this study, 80.95% of study population had severe tubular necrosis, of which 47.61% have<90% TBSA burn and 33.33% have ≥90% TBSA burn. Pearson's correlation coefficient is 0.611 i.e. degree of tubular necrosis is positively correlated with %TBSA burn. The test is statistically significant as the P-value is 0.0368(Fisher's exact test).

Furthermore, 19 (45.24%) patients with presence of tubular necrosis survived for <72 hours and 15 (35.71%) survived for ≥72 hours. Pearson's correlation coefficient is -0.235 i.e. severity of tubular necrosis is negatively correlated with duration of survival. P-value of this test is 0.0043 (chi-square test). Thus, the test is statistically significant. (table 3)

Table 3: Relationship of degree of tubular necrosis with duration of survival.

Degree of Tubular Necrosis	%TBSA		Total n=42	Duration of Survival		Total n=42
	<90%	>90%		<72 hrs	>72 hrs	
severe	20	14	34	19	15	34
Non severe	8	0	8	0	8	08

Discussion

On Gross examination of kidney, there appears to be a small increase in both right and left kidney lengths among individuals who survived more than 24 hours compared to those who died within 24 hours. This may suggest that longer survival allows for less autolysis/shrinkage or reflects better perfusion in earlier stages of systemic failure. However, these differences are minimal (around 1–1.5 mm), so while they might be statistically measurable, they were not statistically significant.

Gross examination of kidneys by Bhetariya BV et al. revealed that most cases (65.63%, 21 out of 32) showed no gross abnormalities. Pale kidneys were observed in 18.75% of cases (6/32), while 15.62% (5/32) presented as heavy and congested. [11]

Surface congestion on gross examination was found to be present in 69.04% cases in the present study along with microscopic finding of interstitial as well as glomerular congestion; however they were not statistically significant when correlated with percentage of TBSA burn and duration of survival.

Histopathological changes in kidney and percentage TBSA burn

Desquamation of the tubular epithelium and presence of tubular casts were the most consistent microscopic changes noted in burn patients with >90% TBSA and were also noted to be consistently present in patients who died within first three days. Furthermore, Presence of interstitial chronic inflammation was found to be positively correlated with duration of survival with P-value of 0.0001(Fisher-exact test) denoting strong statistical significance

In the present study, 59.52% individuals show desquamation of tubular epithelium, out of which 33.33% had > 90% TBSA burn and 26.19% had >60-90% TBSA burn. Desquamation of tubular epithelium is positively correlated with percentage TBSA burn (Pearson's correlation coefficient = 0.766) implying increased desquamation of tubular epithelium with severity of burn which also corroborates with study done by Chakrabarti N et al. [12] Desquamation is a degenerative change of kidney tubules which is found more when the burn affects more body surface area.

Presence of tubular casts was identified in a 54.76% in the present study. Notably, a considerable proportion of these cases with tubular casts involved extensive burns: 14 cases (33.33%) had burns affecting more than 90% TBSA, and an additional 9 cases (21.43%) had burns ranging from 60% to 90% TBSA. Statistical analysis revealed a strong positive correlation (Pearson's r = 0.774) between the presence of tubular casts and the total body surface area of the burn, indicating that the formation of tubular casts is more likely to occur in patients with more extensive burn injuries. Presence of cast is indirect indicator of acute kidney injury. Higher percentage of burn causes reduction of renal perfusion along with release of massive stress related hormones like catecholamines, angiotensin II, aldosterone and vasopressin causing degenerative changes of tubules and finally tubular casts. [13] The findings corroborated well with Mangare et al and Shinde and Keoliya. [4, 14]

Other histopathological findings namely surface congestion, Interstitial congestion and glomerular congestion do show some increasing trend with severity of burn, but the correlation is not statistically significant, possibly due to smaller sample size or plateauing effects.

Histopathological changes in kidney and duration of survival

Both "Desquamation of tubular epithelium" and "Tubular cast" are consistently present in all cases of burn that died within the first 3 days (<24 hours and 1-3 days). This suggests these two kidney findings are likely early and significant responses to severe burn injury that may contribute to early mortality or are indicative of the severity of the initial insult. Shinde and Keoliya found that 31.82% study population had tubular cast of which most of them survived less than 5 days. [14] Similarly, Sevitt et al observed presence of tubular casts 17.44% individuals. [15]

Cloudy Degeneration" and "Interstitial oedema" are observed across all survival timeframes, indicating they might be more general responses to burn injury that can persist or develop at different stages. Agramaso RV observed changes of cloudy swelling in victims who died quickly of suffocation whereas cases showing degenerative changes in the renal tubules survived between 4 and 26 days. [16]

Presence of interstitial chronic inflammation was found to be positively correlated with duration of survival with P-value of 0.0001(Fisher-exact test) denoting strong statistical significance. Inflammation is a complex biological process that plays a critical role in eliminating pathogens and promoting tissue repair after injury. In acute kidney injury (AKI), the intrarenal inflammatory response involves molecular signals released from injured or dying cells, activation of pattern-recognition receptors, recruitment and activation of various resident and circulating immune cell populations, and a coordinated progression from an initial injury-driven inflammatory phase to a later reparative phase. These reparative changes might increase the duration of survival in burn cases. [17] However, in severe burn disease, inflammatory processes in kidneys often result in lethal outcome. Their development may be caused by marked and extended disturbance of nonspecific barrier mechanisms function taking place in severe burn trauma damages, this being an important pathogenetic factor of early bacterial intoxication and sepsis development. One of the leading roles in the development of this process belongs to direct effect of bacteria and product of their vital activity to cellular structure. [18]

Burn injuries commonly lead to Systemic Inflammatory Response Syndrome (SIRS), a condition characterized by immune cell activation and the release of cytokines. This process can culminate in a cytokine storm and subsequent multi-organ

failure. The resulting prolonged pro-inflammatory state also contributes to an increased risk of secondary complications, notably kidney injury, which is recognized as being caused by inflammation. Consequently, interventions aimed at inhibiting inflammation could be beneficial in reducing kidney injury in burn patients. [19, 20]

Degree of Tubular necrosis and its association with %TBSA and duration of survival

The term tubular necrosis is a misnomer, as true cellular necrosis is usually minimal, and the alteration is not limited to the tubular structures. [9] Acute tubular necrosis can occur following ischemia, exposure to toxins, or sepsis and is associated with high morbidity and mortality and is generally most common in hospitalized patients. [3] In this study, 80.95% of study population had severe tubular necrosis, of which 47.61% have<90% TBSA burn and 33.33% have ≥90% TBSA burn. Furthermore, 19 (45.24%) patients with presence of tubular necrosis survived for <72 hours and 15 (35.71%) survived for ≥72 hours.

Extent of burn injury (%TBSA) is positively correlated with the severity of tubular necrosis, and this relationship is statistically significant. Furthermore, the severity of tubular necrosis is negatively correlated with the duration of survival, meaning more severe tubular necrosis is associated with shorter survival times, and this association is also statistically significant. The absence of mortality within the non-severe tubular necrosis group is also a notable finding. Changes of acute tubular necrosis in 81.25% cases were observed by Bhetariya BV et al in their study. [11] Sevitt et al showed that 59% patients with renal tubular necrosis and higher percentage of burn have a stronger association with diffuse tubular necrosis. They also found that 56.86% cases survived for less than 120 hours, among which 49.02% had diffuse and 7.84% had focal tubular necrosis. [15] Sathikumar M also found tubular necrosis among patients who survived for 3 days. [21] Tubular necrosis is a prominent marker for early survival period. During episode of hypovolemia after burn, kidney cannot get enough blood supply and degeneration of epithelium occurs followed by necrosis.

The predominance of ATN in fatal cases suggests that renal injury often develops early and progresses rapidly, frequently becoming irreversible despite supportive care. Clinically, this underscores the importance of early renal-protective interventions in burn patients. Prompt and adequate fluid resuscitation to restore intravascular volume and maintain renal perfusion remains the cornerstone of prevention, while avoiding both under-resuscitation and fluid overload. [22]

Furthermore, the frequent occurrence of severe tubular necrosis supports early identification and aggressive management of contributing factors such as sepsis, prolonged hypotension, and rhabdomyolysis. Early consultation with nephrology and timely initiation of renal replacement therapy in selected patients may mitigate metabolic derangements and prevent further renal injury. Overall, the autopsy findings emphasize that AKI in burn patients is not merely a terminal event but a potentially preventable complication, reinforcing the critical role of early intervention and vigilant renal monitoring in improving outcomes. [23, 24]

The predominance of advanced tubular necrosis suggests that renal injury may progress rapidly to irreversible failure if not addressed early. Clinically, this underscores the importance of prompt and adequate fluid resuscitation, early monitoring of renal function , and avoidance of secondary renal insults . [23, 25]

Limitations

This is a hospital-based study which includes its inherent limitations particularly regarding selection bias, information bias, confounding factors, and generalizability. Further such studies on a large sample size will further help in comprehending the relationship between burn extent, survival duration and renal alteration.

Conclusion

Burn-induced kidney damage manifests in distinct phases. Early on, hypovolemia restricts renal blood supply, causing epithelial degeneration and necrosis, with tubular cast formation and desquamation of tubular epithelium being significant indicators of injury severity and potential early mortality. Simultaneously, an intense pro-inflammatory state exacerbates this damage. In later stages, systemic sepsis becomes a primary driver of organ dysfunction, including kidney failure. Severe burn-related kidney inflammation often leads to death.

Thus, interventions targeting early hypovolemia and inflammation are crucial for protecting kidney function in burn patients. Comprehending the complex interplay between burn severity, survival time, and kidney changes at different levels is vital, necessitating further investigation into the relationship between burn extent, survival duration, and subsequent renal alterations.

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