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Research Article

A Prospective Histopathological Study of Renal Changes in Cases of Flame Burns Brought to Government Medical College Mortuary, Kadapa, Andhra Pradesh

Dr. G. Dinesh[1*], Dr. V. Koteswara Rao[2], Dr. T. Harikrishna[3], Dr. Harish[4]
[1*]Assistant Professor, Dept. of Forensic Medicine and Toxicology, Government Medical College, Kadapa, Andhra Pradesh, India.
[2]Associate Professor, Department of Forensic Medicine and Toxicology, Government Medical College, Nandyal, Andhra Pradesh, India.
[3]Assistant Professor, Department of Forensic Medicine and Toxicology, Government Medical College, Nandyal, Andhra Pradesh, India.
[4]Associate Professor, Dept of Pathology, State Cancer Institute, Kurnool, Andhra Pradesh, India.
Corresponding Author: Dr. G. Dinesh
Assistant Professor, Dept. of Forensic Medicine and Toxicology, Government Medical College, Kadapa, Andhra Pradesh, India.
Email: dineshdinn06@gmail.com
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ABSTRACT
Background: Burn injuries remain a major public health problem and are associated with significant morbidity and mortality worldwide. Acute kidney injury is one of the most important systemic complications following severe flame burns and contributes substantially to mortality. Histopathological examination of renal tissue provides valuable insights into the pathological alterations associated with burn injury and their relationship with survival duration.
Objectives: To identify the histopathological changes in kidneys among fatal flame burn cases and to correlate these findings with post-burn survival.
Materials and Methods: A prospective observational autopsy-based study was conducted in the Department of Forensic Medicine in collaboration with the Department of Pathology, Government Medical College, and Kadapa. Thirty consecutive flame burn fatalities fulfilling the inclusion criteria were enrolled. Following medico-legal autopsy, both kidneys were preserved in 10% neutral buffered formalin, processed routinely, and stained with hematoxylin and eosin for microscopic examination. Histopathological changes were recorded and analyzed using descriptive statistics.
Results: Thirty flame burn cases were included, comprising 15 males and 15 females with a mean age of 43.2 years. Congestion was the most frequent histopathological finding (73.3%), followed by cloudy degeneration (66.7%), acute tubular necrosis of proximal convoluted tubules (63.3%), distal tubular necrosis (46.7%), interstitial inflammatory infiltration (43.3%), tubular casts (20.0%), and pyelonephritis (6.7%). The majority of significant renal changes were observed in patients surviving between 4 and 7 days following burn injury.
Conclusion: Renal histopathological alterations are common following fatal flame burns. Congestion, cloudy degeneration, and acute tubular necrosis constitute the predominant pathological changes. Histopathological evaluation of kidneys provides valuable evidence regarding systemic burn injury and may aid in understanding mechanisms contributing to mortality.

Keywords: Flame burns, Kidney, Histopathology, Acute tubular necrosis, Autopsy, Forensic pathology.

INTRODUCTION

A burn is an injury to the skin or other organic tissue primarily caused by heat or due to radiation, radioactivity, electricity, friction or contact with chemicals. Burns are one of the most devastating injuries a person can sustain. According to a 2018 World Health Organization report, every year, about three lakh people die from burns worldwide, whereas 1,80,000 people die from burn injuries, with the majority living in low- and middle-income countries.[1]

Burns have always remained a serious health problem in India. In 2019, more than 23,000 fire-related deaths were estimated in India, which is about 20% of the global mortality

burden. Additionally, 1·5 million DALYs were attributed to burns.[2] Burns of skin or other tissue are caused by fire, radiant heat, radiation, chemical, or electrical contact. Thermal burns result from heat source (flame, hot liquids, heated solid objects, or hot gases).

Death may occur immediately after burns or may get delayed for days & weeks, where burns may not be the actual cause of death; but its sequels & its complications may result in death.[3] The major cause of death in burn patients includes multiple organ failure and infection but, sometimes the exact cause of death in many fatally burned patients is difficult to detect. Many times, in medico-legal post-mortem examinations in cases of burns, histopathological examination of organs is requested.[4].

Burn injuries are among the most devastating forms of trauma encountered in clinical and forensic practice. They continue to constitute a major cause of accidental morbidity and mortality worldwide, particularly in developing countries where domestic accidents, occupational hazards, and inadequate safety measures remain common.[6] Thermal injuries initiate a complex cascade of inflammatory, metabolic, and hemodynamic responses that affect nearly every organ system. Although extensive skin damage represents the primary injury, systemic complications are largely responsible for adverse clinical outcomes and mortality.[7]

The kidney is invariably involved in burn patients, leading to acute renal failure and other complications. Acute Renal failure (ARF) is a frequent and important phenomenon but not the only one that can induce death.[5]The kidney is one of the earliest organs affected following severe burn injury. Renal dysfunction develops because of hypovolemia, reduced renal perfusion, hemoglobinuria, myoglobinuria, inflammatory mediators, sepsis, and multiorgan dysfunction. Acute tubular injury and subsequent acute kidney injury significantly increase mortality among burn victims. Early recognition of renal involvement remains essential for prompt fluid resuscitation and appropriate supportive management.[5]

Histopathological examination of renal tissue obtained during medico-legal autopsy provides valuable information regarding the sequence of pathological changes occurring after burn injury. Microscopic findings such as cloudy degeneration, vascular congestion, tubular necrosis, interstitial inflammatory infiltration, tubular casts, and pyelonephritis help establish the extent of renal damage and correlate pathological changes with survival duration. Such observations are important not only for understanding the pathophysiology of burn injury but also for medicolegal interpretation of the cause of death.[7,8]

Previous studies have demonstrated that acute tubular necrosis and cloudy degeneration are among the most common renal lesions in fatal burn cases, with their frequency increasing in patients who survive for several days after sustaining burns.[5,7,8] However, published literature from South India remains limited, and regional variations in demographic profile, burn characteristics, and healthcare access may influence histopathological patterns.[9]

Therefore, the present prospective study was undertaken to evaluate renal histopathological changes in fatal flame burn cases brought for medico-legal autopsy at Government Medical College, Kadapa, and to correlate these findings with post-burn survival duration.

MATERIAL AND METHODS

Study design and setting:

A prospective observational autopsy-based study was conducted in the Department of Forensic Medicine in collaboration with the Department of Pathology, Government Medical College, Kadapa, and Andhra Pradesh. The study included medicolegal autopsies of fatal flame burn cases received at the Government Medical College Mortuary. The study protocol specified a prospective design, histopathological examination of both kidneys using routine H&E staining, and a planned sample size of 30 cases.

Study duration: The study was conducted over one year after obtaining approval from the Institutional Ethics Committee.

Study population: Thirty consecutive medico-legal autopsy cases of fatal flame burns fulfilling the inclusion criteria were included.

Inclusion criteria

- Fatal cases of flame burns.
- Cases with varying post-burn survival duration.

Exclusion criteria

- Burns due to causes other than flame burns.
- Decomposed bodies.

Data collection: Demographic information, burn percentage, duration of survival, and relevant medico-legal details were collected from hospital records, police documents, inquest reports, and post-mortem records. During autopsy, both kidneys were removed, fixed in 10% neutral buffered formalin, routinely processed, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Histopathological examination assessed cloudy degeneration, congestion, acute tubular necrosis of proximal and distal convoluted tubules, tubular casts, pyelonephritis, and interstitial inflammatory infiltration.

The study was approved by Institution Ethics Committee of Government Medical College, Kapada (IEC. No 2/GMC/KDP/2024 dated 02.04.2024)

Statistical analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages.

RESULTS

Thirty flame burn autopsy cases were included in the study. The study population comprised 15 (50.0%) males and 15 (50.0%) females. The mean age of the study population was 43.2 years.

Table 1. Demographic Profile of Study Population (N=30)

Variable	Number (%)
Male	15 (50.0)
Female	15 (50.0)
Total	30 (100)
Mean age	43.2±11.25 years

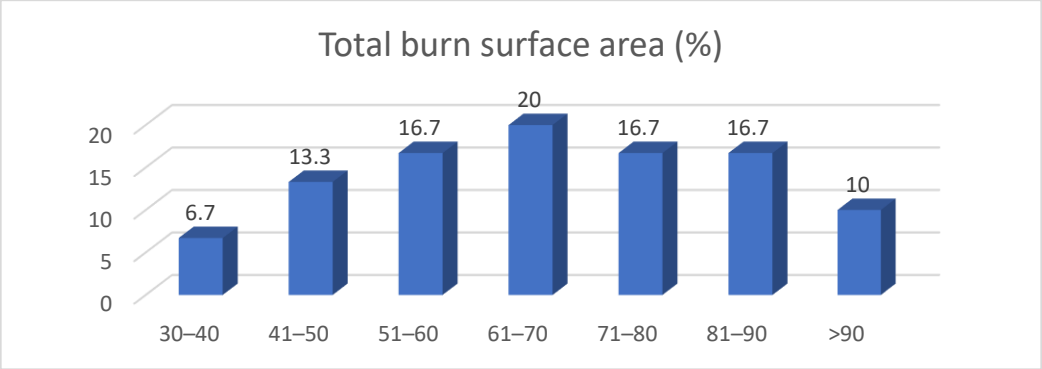

Figure 1. Distribution of Fatal Flame Burn Cases According To Total Burn Surface Area (N = 30)

The majority of the victims sustained burns involving 61–70% of the total body surface area (20.0%), followed by 51–60%, 71–80%, and 81–90% burns (16.7% each). Only 6.7% of cases had burns involving 30–40% of the body surface area.

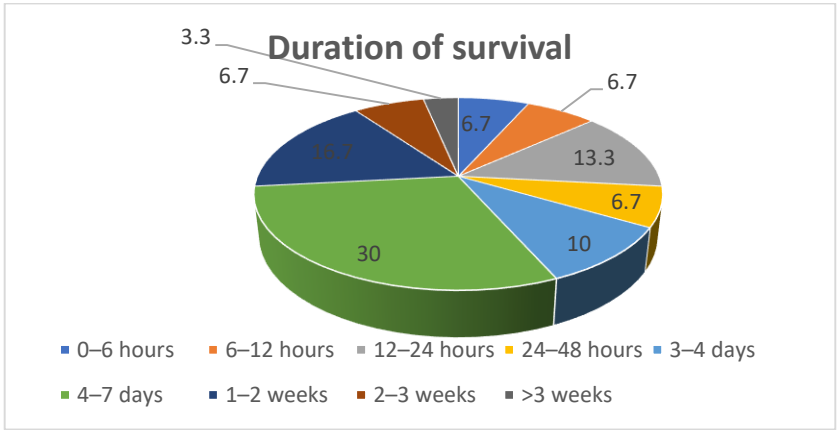

Figure 2. Distribution of Fatal Flame Burn Cases According To Duration of Survival (N = 30)

Most burn victims survived for 4–7 days (30.0%), followed by 1–2 weeks (16.7%) and 12–24 hours (13.3%). Survival beyond three weeks was observed in only one (3.3%) case

Histopathological findings

Congestion was the commonest renal histopathological alteration, observed in 22 (73.3%) cases, followed by cloudy degeneration in 20 (66.7%) cases. Acute tubular necrosis involving the proximal convoluted tubules was present in 19 (63.3%) cases, while distal tubular necrosis was identified in 14 (46.7%) cases. Interstitial inflammatory infiltration was observed in 13 (43.3%) cases. Tubular casts were present in six (20.0%) cases and pyelonephritis was noted in two (6.7%) cases.

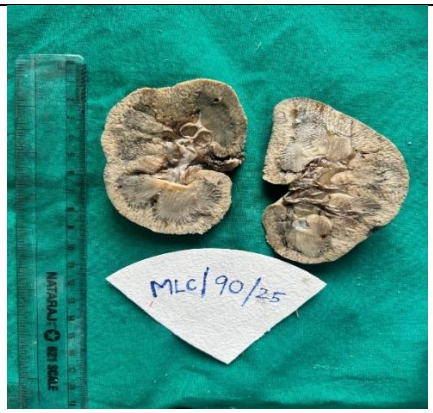

Figure 3: Enlarged, Congested And Pale

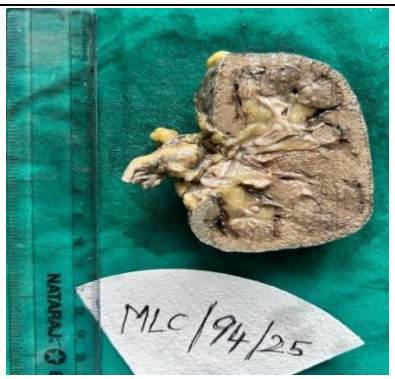

Figure 4: Cortico-Medullary Distinction Cannot Be Clearly Demarcated

Figure 5: Congested

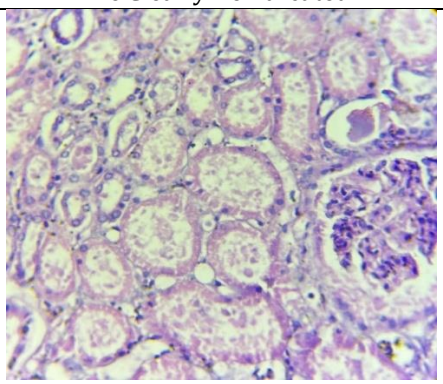
Figure 6: Showing Tubular Necrosis

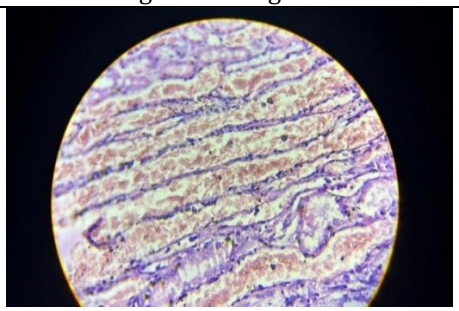
Figure 7: Interstitium Showing Moderate Inflammatory Infiltrate With Hemorrhage

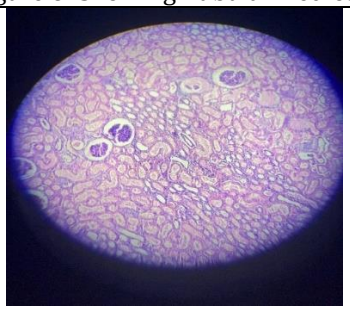
Figure 8: Blood Vessels Are Dilated And Congested

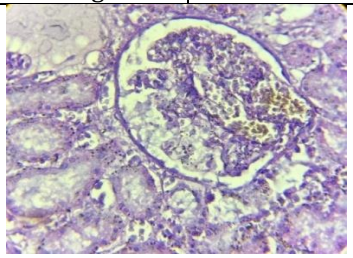
Figure 9: Glomerulus –Showing Mild Hemorrhage

Table 2. Renal Histopathological Findings (N=30)

Histopathological finding	Frequency	Percentage
Congestion	22	73.3
Cloudy degeneration	20	66.7
ATN (Proximal tubules)	19	63.3
ATN (Distal tubules)	14	46.7
Interstitial infiltration	13	43.3
Tubular casts	6	20.0
Pyelonephritis	2	6.7

Table 3. Distribution of Renal Histopathological Changes According To Duration of Survival (N=30)

Histopathological finding	0–6 h	6–12 h	12–24 h	24–48 h	3–4 days	4–7 days	1–2 weeks	2–3 weeks	>3 weeks	Total
Cloudy degeneration	2	1	3	1	2	8	1	1	1	20 (66.7)
Congestion	1	2	3	1	3	7	3	1	1	22 (73.3)
ATN (PCT)	1	0	2	2	1	8	4	0	1	19 (63.3)
ATN (DCT)	0	2	0	2	2	5	3	0	0	14 (46.7)
Tubular casts	0	0	1	1	1	3	0	0	0	6 (20)
Pyelonephritis	0	0	0	0	0	1	0	1	0	2 (6.7)
Interstitial inflammatory infiltration	0	2	1	1	3	4	1	1	0	13 (43.3)

Most renal histopathological alterations were observed among patients who survived 4-7 days after sustaining flame burns. Congestion (7 cases), cloudy degeneration (8 cases), and Proximal tubular acute tubular necrosis (8 cases) were the predominant findings during this survival period, indicating progressive ischemic and inflammatory renal injury.

Table 4. Distribution of Cases According To Cause of Death and Renal Histopathological Changes (N = 30)

Histopathological finding	Septicemia (n)	Shock (n)	Toxaemia (n)	Total
Cloudy degeneration	7	6	7	20 (66.7)
Congestion	8	6	8	22 (73.3)
Acute tubular necrosis (PCT)	7	5	7	19 (63.3)
Acute tubular necrosis (DCT)	8	3	3	14 (46.7)
Tubular casts	2	1	3	6 (20)
Pyelonephritis	0	1	1	2 (6.7)
Interstitial inflammatory infiltration	4	4	5	13 (43.3)

Acute renal histopathological alterations were observed across all three causes of death. Congestion was the most frequent finding in cases of septicemia (8 cases) and toxaemia (8 cases), whereas six cases with shock demonstrated vascular congestion. Cloudy degeneration was equally distributed between septicemia and toxaemia (7 cases each) and was observed in six cases of shock. Acute tubular necrosis involving the proximal convoluted tubules was present in seven cases

Each of septicemia and toxaemia and in five cases of shock. Distal tubular necrosis was predominantly associated with septicemia (8 cases), while tubular casts and pyelonephritis were comparatively infrequent. Interstitial inflammatory infiltration was identified in five cases of toxaemia and four cases each of septicemia and shock.

These findings indicate that congestion, cloudy degeneration, and acute tubular necrosis were

the predominant renal lesions irrespective of the cause of death, with septicemia showing the greatest burden of advanced tubular injury.

DISCUSSION

The present prospective autopsy-based study evaluated the renal histopathological changes in 30 fatal flame burn cases. Renal involvement following severe thermal injury is a well-recognized complication and contributes significantly to burn-related mortality through acute kidney injury and multiorgan dysfunction. In the present study, congestion (73.3%), cloudy degeneration (66.7%), and acute tubular necrosis (ATN) of the proximal convoluted tubules (63.3%) were the predominant histopathological findings. These observations suggest that renal ischemia, hypovolemia, systemic inflammatory response, and sepsis play important roles in the pathogenesis of renal injury following severe burns.

In the present study, males and females were equally affected (50% each), with a mean age of 43.2 years. Although several Indian studies have reported a female predominance among burn fatalities due to domestic cooking accidents, the equal gender distribution observed in the present study may reflect regional variations in the epidemiology of flame burns. Mangare and Punia reported a female predominance (55%) among burn victims, with the majority belonging to the 20–40-year age group.[5] Similarly, Shinde and Keoliya also observed a higher proportion of females among fatal burn cases.1[0]These differences may be attributed to variations in demographic characteristics, cultural practices, and referral patterns across different regions.

The majority of victims in the present study sustained burns involving 61–70% of the total body surface area, while most patients survived for 4–7 days following injury. This finding is consistent with the observations of Mangare and Punia, who reported that most fatalities had burns involving more than 50% of the total body surface area, with the highest mortality occurring between 4 and 7 days after injury.[5] The higher frequency of deaths during this period is likely related to the development of systemic inflammatory response syndrome, septicemia, and progressive multiorgan dysfunction after the initial burn shock.

Congestion was the most frequent renal histopathological alteration observed in the present study (73.3%). Similar findings were reported by Mangare and Punia[5], who documented congestion in 66.25% of cases, and by Shinde and Keoliya[10], who also identified vascular congestion as one of the common microscopic findings in fatal burn cases. Renal congestion is considered an early manifestation of circulatory disturbances resulting from hypovolemia, increased vascular permeability, and inflammatory mediator release following severe burns.[11]

Cloudy degeneration was identified in 66.7% of the present cases and represented one of the earliest microscopic changes in renal tubular epithelial cells. Mangare and Punia[5] reported cloudy degeneration in 75% of their cases, whereas Bhetariya et al.[12] observed cloudy swelling in a smaller proportion of burn fatalities. The slightly lower frequency in the present study may be related to differences in burn severity, duration of survival, and sample size. Cloudy degeneration is generally regarded as an early reversible manifestation of cellular injury resulting from hypoxia and metabolic derangement.[12]

Acute tubular necrosis of the proximal convoluted tubules was observed in 63.3% of cases, while distal tubular necrosis was present in 46.7%. These findings are comparable with those reported by Mangare and Punia[5], who found proximal and distal tubular necrosis in 52.5% and 51.3% of cases, respectively. Sevitt also described proximal and distal tubular necrosis as characteristic renal lesions in patients dying from severe burns. Acute tubular necrosis develops primarily due to prolonged renal hypoperfusion, shock, hemoglobinuria, myoglobinuria, and sepsis and remains one of the principal pathological substrates of acute kidney injury in burn patients.[13]

Interstitial inflammatory infiltration was observed in 43.3% of the present cases. Mangare and Punia[5] reported lymphocytic infiltration in 36.25% of burn fatalities, suggesting that inflammatory changes become increasingly prominent with longer survival. Persistent systemic inflammation following burns contributes to progressive renal tissue damage and eventually predisposes patients to multiorgan failure.

Tubular casts were present in 20% of the cases, whereas pyelonephritis was identified in only 6.7%. Similar observations have been reported in previous autopsy studies, indicating that these lesions are relatively uncommon and usually occur in patients

surviving long enough to develop secondary infective complications. The low frequency of pyelonephritis in the present study may be attributable to early mortality in a substantial proportion of patients.

The association between renal histopathological findings and duration of survival demonstrated that most significant lesions occurred among patients surviving 4–7 days after burn injury. Similar observations were reported by Mangare and Punia[5], who concluded that cloudy degeneration, congestion, and acute tubular necrosis were most pronounced during this survival period. This finding emphasizes the importance of aggressive fluid resuscitation, hemodynamic stabilization, and prevention of sepsis during the first week after burn injury to reduce renal complications.

The present study also evaluated the relationship between cause of death and renal histopathological changes. Although congestion and acute tubular necrosis were observed across all categories of death, no statistically significant association was demonstrated between cause of death and individual histopathological lesions. This finding suggests that renal injury develops through common pathophysiological mechanisms irrespective of the immediate terminal event and is primarily influenced by burn severity and systemic inflammatory response.

Overall, the findings of the present study are consistent with previously published autopsy studies and reinforce the concept that congestion, cloudy degeneration, and acute tubular necrosis represent the hallmark renal histopathological changes following fatal flame burns. Histopathological examination of kidneys during medicolegal autopsy not only assists in understanding the mechanisms of burn-related mortality but also provides valuable information regarding the progression of renal injury. Early recognition and appropriate management of burn shock, adequate fluid resuscitation, and prompt treatment of sepsis remain essential strategies for minimizing renal damage and improving survival in patients with severe flame burns.

The present study has several strengths. It was designed as a prospective autopsy-based investigation, enabling systematic collection of data and minimizing retrospective bias. Histopathological examination of renal tissue provided direct microscopic confirmation of renal lesions associated with fatal flame burns. Furthermore, all tissue specimens were processed using standardized histopathological techniques, ensuring uniformity in tissue fixation, processing, staining, and microscopic evaluation. The inclusion of consecutive flame burn cases also reduced selection bias and enhanced the representativeness of the study population.

Despite these strengths, the study has certain limitations. The relatively small sample size of 30 cases and the single-centre study design may limit the generalizability of the findings to the wider population. Additionally, biochemical indicators of renal function, such as serum creatinine and blood urea nitrogen, could not be correlated with the histopathological findings because such data were unavailable for all cases. Long-term clinical follow-up before death was also not feasible, restricting assessment of the progression of renal injury and treatment response.

Future studies should include larger multicentre cohorts to improve the external validity of the findings. Correlating renal histopathological alterations with biochemical markers of acute kidney injury, including serum creatinine and novel biomarkers, would provide a better understanding of clinicopathological relationships. Advanced techniques such as immunohistochemistry and molecular pathology may further elucidate the mechanisms of early renal injury following severe flame burns. In addition, the development of prognostic models integrating burn surface area, duration of survival, clinical parameters, and histopathological findings may help predict outcomes and guide the management of patients with severe burn injuries.

CONCLUSION

Renal involvement is a frequent pathological consequence of fatal flame burns. Congestion, cloudy degeneration, and acute tubular necrosis were the most common histopathological abnormalities identified in the present study. These findings emphasize the importance of early fluid resuscitation, prevention of renal ischemia, and prompt management of burn patients to reduce renal complications and mortality. Histopathological examination of kidneys during medico-legal autopsy remains valuable for establishing burn-related systemic organ injury and understanding the mechanisms of death.

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