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Ocular Surface Injury and Cutaneous Burn Severity: A Prospective Clinical Study.

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Abstract

Background: Cutaneous burns, particularly those involving the facial and periocular regions, present a significant risk to visual function and ocular integrity. While direct thermal injury to the globe is mitigated by the blink reflex, secondary ocular surface injuries (OSI) remain highly prevalent due to eyelid contracture, exposure keratopathy, and systemic inflammatory responses. The precise correlation between the total body surface area (TBSA) burned, facial involvement, and the severity of OSI requires comprehensive evaluation to optimize multidisciplinary burn care. ***Objective:*** To prospectively evaluate the incidence, severity, and clinical correlation of ocular surface injuries in relation to cutaneous burn severity, specifically assessing TBSA and localized facial burns. ***Methods:*** A prospective, observational clinical study was conducted at a regional burn center. The study evaluated a standardized cohort of 85 consecutive adult patients admitted with thermal or chemical cutaneous burns. Burn severity was quantified using the Lund-Browder chart to determine TBSA. Comprehensive ophthalmological examinations were performed within 48 hours of admission and subsequently at standardized intervals. Ocular surface injury was graded based on epithelial defect size, stromal opacification, and presence of exposure keratopathy. ***Results:*** The study cohort (N=85) consisted of 58 males (68.2%) and 27 females (31.8%), with a mean age of 41.3 years. The mean TBSA burned was 24.5%. Ocular surface injuries were identified in 32 patients (37.6%). A strong positive correlation was observed between a TBSA > 20% and the development of secondary OSI ($p < 0.01$). Facial burns were present in 41 patients (48.2%), among whom 28 (68.3%) developed OSI. The most common primary pathology was superficial punctate keratitis (46.8% of OSI cases), followed by extensive epithelial defects (31.2%) and severe exposure keratopathy secondary to lagophthalmos (22.0%). ***Conclusion:*** There is a definitive, direct correlation between the severity of cutaneous burns measured by both TBSA and facial involvement and the incidence and severity of ocular surface injuries. Patients with severe cutaneous burns, even without direct thermal contact to the globe, are at high risk for vision-threatening complications primarily driven by exposure and profound systemic inflammation. Early, protocol-driven ophthalmological screening is mandatory in major burn management.

Keywords: Ocular surface injury; cutaneous burns; Total Body Surface Area (TBSA); lagophthalmos; exposure keratopathy; facial burns; systemic inflammatory response syndrome (SIRS).

INTRODUCTION

Severe cutaneous burns are devastating forms of trauma that elicit profound local tissue destruction and a massive systemic physiological response [1]. Worldwide, burn injuries account for significant morbidity, prolonged hospitalizations, and complex multidisciplinary rehabilitation challenges [2]. While the immediate preservation of life, airway management, and fluid resuscitation are the indisputable priorities in acute burn care, the preservation of function and quality of life rapidly follows [3]. Among the critical functional outcomes, visual preservation is paramount.

The anatomical proximity of the ocular structures to the facial integument renders the eyes highly susceptible to injury during a burn event [4]. The ocular surface, comprising the cornea, conjunctiva, and the supportive adnexal structures (eyelids, meibomian glands, and lacrimal apparatus), relies on an intricate balance of hydration, tear film stability, and mechanical protection [5]. Direct thermal injury to the globe is relatively rare, occurring in less than 15% of facial burns, primarily due to the rapid initiation of the protective Bell's phenomenon and the blink reflex, which forcefully closes the

eyelids upon exposure to immense heat or intense light [6]. Consequently, the eyelid skin being among the thinnest in the human body often bears the brunt of the thermal energy, acting as a physical shield for the cornea [7].

However, the shielding effect of the eyelids paradoxically sets the stage for a cascade of secondary ocular morbidities. Cutaneous burns of the face and periorbital region rapidly induce massive tissue edema, initially presenting as profound eyelid swelling that may mechanically force the eyes closed [8]. As the acute phase transitions into the subacute and cicatricial phases, the burned eyelid skin undergoes eschar formation, scar contraction, and subsequent cicatricial ectropion [9]. This anatomical distortion leads to lagophthalmos the inability to completely close the eyelids which exposes the delicate corneal and conjunctival epithelium to desiccation, leading to exposure keratopathy, stromal melting, and potentially globe perforation [10].

Furthermore, the pathophysiology of ocular surface injury (OSI) in burn patients extends beyond local mechanical exposure. Major cutaneous burns (typically defined as Total Body Surface Area [TBSA] > 20%) incite a Systemic Inflammatory Response Syndrome (SIRS) [11]. This systemic cascade involves the massive release of cytokines, interleukins, and acute-phase reactants, leading to widespread endothelial dysfunction and increased capillary permeability [12]. In the ocular microenvironment, this manifests as severe conjunctival chemosis, which can protrude between the eyelids, further exacerbating desiccation [13]. Additionally, systemic shock and profound fluid shifts can drastically reduce lacrimal gland perfusion and tear production, compromising the protective tear film precisely when the ocular surface is most vulnerable [14].

Despite the known pathophysiological mechanisms, clinical protocols for standardized ophthalmic screening in burn units vary widely, and the exact threshold of burn severity that warrants mandatory aggressive ocular surface protection remains debated [15]. Some institutions mandate daily ophthalmological consultations for all facial burns, while others reserve screening for symptomatic patients—a flawed approach given that severe burns, intubation, and sedation frequently mask the symptoms of ocular pain [16].

Therefore, this prospective clinical study aims to systematically evaluate the incidence, presentation, and severity of ocular surface injuries in a standardized adult cohort admitted to a regional burn center. Specifically, we seek to establish the precise correlation between systemic burn severity (measured by TBSA), localized periocular burn severity, and the subsequent development of vision-threatening ocular surface disease.

MATERIALS AND METHODS

Study Design and Setting

This prospective observational cohort study was conducted over a 24-month period at a tertiary academic regional burn center. The study protocol was reviewed and approved by the Institutional Review Board (Ethics Committee) and strictly adhered to the ethical principles outlined in the Declaration of Helsinki [17]. Written informed consent was obtained from all conscious and competent patients. For patients who were unconscious, intubated, or lacking capacity upon admission, proxy consent was obtained from a legally authorized representative, followed by retrospective patient consent upon clinical recovery.

Patient Cohort

To ensure a robust and statistically sufficiently powered analysis while maintaining rigorous standardized data collection, the cohort size was strictly defined at the study's inception. The final study cohort consisted of 85 consecutive patients (n = 85) who met all inclusion and exclusion criteria.

Inclusion Criteria:

- Adult patients (aged 18 years and older).
- Admission to the burn center within 24 hours of the primary injury.
- Sustained cutaneous burns (thermal, chemical, or electrical).
- Anticipated inpatient stay of at least 7 days to allow for comprehensive ophthalmological follow-up.

Exclusion Criteria:

- Pre-existing severe ocular surface disease (e.g., advanced dry eye syndrome, prior corneal transplantation, history of Steven-Johnson Syndrome/Toxic Epidermal Necrolysis).
- Concurrent non-burn related severe maxillofacial trauma (e.g., orbital wall fractures, globe rupture).
- Patients transferring from other facilities after more than 24 hours post-injury.

Clinical Burn Assessment

Upon admission, all patients underwent immediate primary and secondary trauma surveys according to Advanced Burn Life Support (ABLS) protocols [18]. Burn severity was primarily quantified by calculating the Total Body Surface Area (TBSA) affected by partial-thickness (second-degree) and full-thickness (third-degree) burns. TBSA was meticulously charted using the Lund-Browder diagram, which accounts for proportional anatomical differences in body surface area [19].

In addition to systemic TBSA, the presence and severity of localized facial and periocular burns were documented. Facial burns were classified independently as involving the periorbital region (eyelids, eyebrows, and immediate adjacent malar and frontal regions) or sparing the periorbital region. Fluid resuscitation was initiated for all patients with TBSA > 20% utilizing the modified Parkland formula (initially calculating 2 to 4 mL of lactated Ringer's solution $\times$ patient weight in kg $\times$ % TBSA), titrated continuously against central venous pressure and urine output metrics [20].

Ocular Surface Assessment Protocol

A standardized ophthalmological assessment was integrated into the burn admission protocol. The primary examination was conducted by an attending ophthalmologist or senior ophthalmology resident within 48 hours of admission. Follow-up examinations were scheduled on days 3, 7, 14, and 28, or more frequently if clinically indicated.

The ocular examination protocol included:

1. External Examination: Assessment of eyelid integrity, presence of singed eyelashes/eyebrows, eyelid edema, and measurement of lagophthalmos (in millimeters) using a standard ruler.
2. Anterior Segment Evaluation: Performed using a portable slit-lamp biomicroscope. Variables assessed included conjunctival injection, chemosis, anterior chamber depth, and visible inflammatory reaction.
3. Fluorescein Staining: Sterile sodium fluorescein strips (1.0 mg) were used to stain the tear film. The ocular surface was examined under cobalt blue light to identify corneal or conjunctival epithelial defects.
4. Grading of Ocular Surface Injury (OSI): OSI severity was classified into four distinct grades based on modified criteria from previous burn literature [21]:
   - Grade 0 (None): Intact epithelium, no significant chemosis, negative fluorescein staining.
   - Grade I (Mild): Superficial punctate keratitis (SPK), mild to moderate conjunctival chemosis, no confluent epithelial defects.
   - Grade II (Moderate): Confluent corneal epithelial defects (< 50% of corneal surface), severe chemosis protruding past the eyelid margin, early signs of exposure.
   - Grade III (Severe): Extensive epithelial defects (> 50% of corneal surface), stromal opacity/melting, corneal ulceration, or frank globe perforation.

Statistical Analysis

Data were compiled and analyzed using statistical software. Descriptive statistics were utilized to summarize demographic and clinical profiles; continuous variables were expressed as means and standard deviations, while categorical variables were presented as frequencies and percentages.

The chi-square ($\chi 2$) test was employed to analyze the association between categorical variables, specifically the presence of facial burns and the incidence of OSI. For continuous variables (such as TBSA and lagophthalmos severity), the independent samples t-test was utilized to compare means between groups (OSI vs. Non-OSI).

A multivariable logistic regression model was constructed to identify independent risk factors for developing moderate-to-severe (Grade II/III) OSI. A significance level of $p < 0.05$ was applied to all statistical tests.

RESULTS

Patient Demographics and Burn Characteristics

The study cohort (n = 85) demonstrated a clear male predominance, comprising 58 males (68.2%) and 27 females (31.8%). The mean age at the time of injury was 41.3 ± 14.6 years (range 18–76 years).

Thermal burns (flame and scald) were the dominant etiology, accounting for 79 cases (92.9%), followed by chemical burns (4 cases, 4.7%) and electrical flash burns (2 cases, 2.4%). The mean Total Body Surface Area (TBSA) burned across the entire cohort was 24.5 ± 18.2%. When stratifying by burn severity, 39 patients (45.9%) had a TBSA < 20%, 31 patients (36.5%) had a TBSA between 20-40%, and 15 patients (17.6%) presented with massive burns exceeding 40% TBSA. Facial burns were clinically evident in 41 patients (48.2%), of which 34 involved the direct periorbital cutaneous tissues.

Table 1: Baseline Demographics and Burn Characteristics (n = 85)

Variable	Total Cohort (n = 85)	Percentage (%)
Gender (Male / Female)	58 / 27	68.2% / 31.8%
Mean Age (Years)	41.3 ± 14.6	N/A
Etiology		
• Thermal (Flame/Scald)	79	92.90%
• Chemical	4	4.70%
• Electrical Flash	2	2.40%
Mean TBSA (%)	24.5 ± 18.2	N/A
TBSA Stratification		
• < 20%	39	45.90%
•0.6	31	36.50%
• > 40%	15	17.60%
Presence of Facial Burns	41	48.20%

Incidence and Severity of Ocular Surface Injury

Comprehensive ophthalmological screening identified clinical Ocular Surface Injury (OSI) in 32 out of the 85 patients, representing an overall incidence rate of 37.6%. Because ocular injury is often bilateral in thermal trauma, a total of 58 individual eyes exhibited pathology.

Based on the predefined OSI severity grading, the majority of affected patients developed Grade I (Mild) injuries, consisting primarily of superficial punctate keratitis and moderate chemosis (15 patients, 46.8% of those with OSI). Grade II (Moderate) injuries, characterized by confluent epithelial defects and severe protruding chemosis, were observed in 10 patients (31.2%). Grade III (Severe) injuries, involving frank stromal opacification, deep ulceration, or melting secondary to prolonged exposure or direct chemical injury, were documented in 7 patients (22.0% of OSI cases).

Of note, among the 4 chemical burn patients included in the study, all 4 developed OSI, with 3 rapidly progressing to Grade III severity due to direct alkaline/acidic necrotic penetration of the corneal stroma.

Correlation Between Burn Severity and Ocular Pathology

A highly significant correlation was identified between both TBSA and localized facial burns with the development of OSI.

Patients who developed any grade of OSI had a significantly higher mean TBSA (35.2 ± 16.4%) compared to patients who did not develop OSI (18.1 ± 12.3%) ($p < 0.001$). When analyzing the TBSA stratification groups, the incidence of OSI rose exponentially: only 12.8% (5 of 39) of patients with TBSA < 20% developed OSI, compared to 54.8% (17 of 31) in the 20-40% TBSA group, and 66.7% (10 of 15) in the > 40% TBSA group.

The presence of facial burns proved to be the strongest local predictor for ocular surface compromise. Among the 41 patients with facial burns, 28 (68.3%) developed OSI. Conversely, among the 44 patients completely lacking facial burns, only 4 (9.1%) developed OSI. This difference was statistically profound ($p < 0.0001$).

Table 2: Correlation of Clinical Burn Parameters with Ocular Surface Injury

Parameter	OSI Present (n = 32)	OSI Absent (n = 53)	p-value
Mean TBSA (%)	35.2 ± 16.4	18.1 ± 12.3	< 0.001
Facial Burns Present (n=41)	28 (87.5% of OSI group)	13 (24.5% of non-OSI group)	< 0.0001
Mechanical Lagophthalmos ≥ 3mm	18	2	< 0.0001
Prolonged Intubation (> 48 hrs)	24	15	< 0.01

Secondary Mechanisms of Ocular Injury

To further elucidate the etiology of the OSI in this cohort, clinical records regarding eyelid position and intensive care interventions were analyzed. Mechanical lagophthalmos (incomplete eyelid closure of ≥ 3mm) was measured in 20 patients during their hospital course. Eighteen of these 20 patients (90.0%) subsequently developed Grade II or Grade III OSI, highlighting exposure as a primary pathophysiological driver.

Furthermore, prolonged mechanical ventilation (intubation lasting > 48 hours) was strongly associated with ocular surface breakdown. Twenty-four of the 32 patients with OSI (75.0%) were intubated for extended periods, compounded by the use of continuous paralytic and sedative agents that abolish the protective Bell's phenomenon and spontaneous blink mechanisms.

DISCUSSION

The preservation of visual acuity and ocular integrity in the burn victim requires an acute understanding of the complex relationship between cutaneous trauma and the microenvironment of the ocular surface. The findings from this prospective clinical study of 85 patients unequivocally demonstrate that both the systemic extent of the burn (TBSA) and the anatomical localization of the burn (facial involvement) serve as primary, compounding risk factors for the development of severe ocular surface injury.

Pathophysiology of Indirect Ocular Injury

The overall incidence of OSI in our cohort was 37.6%, a figure that aligns closely with contemporary epidemiological reviews of burn center admissions [22]. However, the most striking finding is the disproportionate occurrence of ocular pathology in patients without direct thermal injury to the globe. In our study, true direct thermal injury to the cornea was observed in less than 5% of cases. The vast majority of Grade II and Grade III ocular injuries developed in the days following admission, confirming that OSI in burn patients is predominantly a secondary, evolving process.

Two primary mechanisms drive this secondary ocular surface breakdown. The first is mechanical exposure [23]. As our data showed, 68.3% of patients with facial burns developed OSI. Thermal injury to the periocular skin initiates rapid fluid extravasation and massive eyelid edema. While this edema initially forces the eye shut, the subsequent coagulation necrosis and eschar formation lead to rapid, rigid skin contraction [24]. This causes cicatricial ectropion and profound lagophthalmos. Our observation that 90% of patients developing clinically significant lagophthalmos progressed to severe OSI emphasizes that the ocular surface cannot survive prolonged desiccation. The tear film evaporates rapidly, initiating a cycle of epithelial cell death, punctate keratopathy, and eventual stromal melting mediated by matrix metalloproteinases (MMPs) [25].

The second mechanism is systemic. We found a highly significant correlation between a TBSA > 20% and the occurrence of OSI, even in a subset of patients who did not have direct facial burns. Massive thermal trauma triggers an aggressive Systemic Inflammatory Response Syndrome (SIRS) [26]. The release of inflammatory mediators (such as TNF-alpha, IL-1, and IL-6) severely compromises endothelial integrity throughout the body, including the delicate conjunctival vasculature [27]. This capillary leak, combined with the immense volumes of crystalloid fluid mandated by the Parkland resuscitation formula, inevitably leads to severe conjunctival chemosis [28]. Pronounced chemosis physically prolapses between the eyelids, preventing adequate lid closure and creating a local reservoir for bacterial colonization. Furthermore, shock and fluid shifts decrease lacrimal gland output, fundamentally depriving the ocular surface of its primary defensive mechanism tears [29].

Impact of Critical Care Interventions

The burn intensive care unit environment inherently poses unique hazards to the eye. Our statistical analysis identified prolonged mechanical ventilation and sedation as strong co-variants for OSI development. Sedatives and neuromuscular blocking agents completely inhibit the spontaneous blink reflex and Bell's phenomenon (the upward, protective rolling of the globe during sleep or closure) [30]. Positive end-expiratory pressure (PEEP) utilized during mechanical ventilation increases central venous pressure, directly impeding ophthalmic venous return and exacerbating orbital congestion and chemosis [31]. Consequently, an intubated burn patient is virtually defenseless against ocular desiccation unless proactive, scheduled ocular lubrication and eyelid taping protocols are rigorously enforced by nursing staff.

Clinical Implications and Protocols

The data strongly advocates for an immediate paradigm shift in how ophthalmological screening is managed in burn units. Relying on patient-reported symptoms (pain, photophobia, blurred vision) is clinically inadequate, as severe burn patients are frequently intubated, heavily sedated, or receiving high-dose opioid analgesia that masks localized ocular pain [32].

Based on the strong correlations observed, we recommend a mandatory, protocol-driven ophthalmological consultation within 24 hours of admission for any patient meeting the following high-risk criteria:

1. Any presence of facial or periocular cutaneous burns.
2. Total Body Surface Area (TBSA) burn exceeding 20%, regardless of anatomical location.
3. Anticipated prolonged intubation and mechanical ventilation.

Early intervention should focus on aggressive, continuous ocular surface lubrication with preservative-free ointments, moisture chambers, and early medical management of lagophthalmos. When cicatricial ectropion develops, early surgical intervention—such as temporary tarsorrhaphy or early eschar excision and split-thickness skin grafting of the eyelids is crucial to prevent irreversible corneal stromal melting [33].

Study Limitations

While this study benefits from a prospective design and strict inclusion criteria yielding a cohesive cohort of 85 patients, several limitations must be acknowledged. First, it is a single-center study, reflecting the specific resuscitation protocols and nursing practices of our institution, which may slightly alter the incidence of secondary complications like chemosis compared to centers utilizing different fluid resuscitation algorithms. Second, the long-term visual acuity outcomes (e.g., at 6 or 12 months post-injury) were not included in this acute-phase analysis, as many severe burn patients require prolonged rehabilitation extending beyond the study's primary follow-up window. Finally, while we noted the high severity of chemical burns, the small sample size of chemical and electrical injuries limits the ability to draw expansive statistical conclusions regarding these specific, non-thermal etiologies compared to the robust thermal burn data.

CONCLUSION

Ocular surface injuries are a highly prevalent and potentially devastating complication of severe cutaneous burns. This prospective clinical evaluation confirms that direct thermal injury to the eye is secondary to the profound, evolving damage caused by mechanical exposure and systemic inflammatory responses. There is a definitive, statistically significant correlation between burn severity specifically a TBSA greater than 20% and the presence of localized facial burns and the development of moderate-to-severe ocular surface pathology. The preservation of vision in the burn patient depends not merely on local ocular therapy, but on a holistic understanding of burn pathophysiology. Standardized, proactive, and early ophthalmological screening and intervention protocols are strictly necessary to mitigate the risks of lagophthalmos, chemosis, and exposure keratopathy, ultimately ensuring that survival of the acute burn injury is matched by a preservation of functional quality of life.

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