



Original Research Article

CARBIDE GUN-INDUCED OCULAR TRAUMA: AN EMERGING CAUSE OF PREVENTABLE VISUAL MORBIDITY

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ABSTRACT

Background: Aim: Carbide gun-induced ocular trauma has emerged as a distinct and preventable pattern of thermochemical eye injury in India. The present review was prepared to synthesize the available evidence on epidemiology, mechanisms, clinical profile, management, outcomes, and prevention of carbide gun-related eye injuries, while comparing the present institutional dataset with the findings of the published studies. A secondary aim was to clarify whether our cases reflect the same trend described in recent Indian literature, namely unilateral severe injuries associated with direct inspection of a malfunctioning gun, limbal ischemia, corneal haze, epithelial defects, and persistent visual morbidity despite treatment.

Materials and Methods: This systematic-style review was based on our center's analysis, and compares it with a public health editorial on carbide gun-associated ocular injuries, one case-series report of five children and young adults from central India, one retrospective analysis of 28 eyes with calcium carbide-related thermochemical injury and one ophthalmic perspective article. The available studies were reviewed for patient demographics, setting of injury, mechanism, laterality, presenting visual acuity, ocular findings, acute management, surgical intervention, complications, and visual outcomes. Data from the spreadsheet were interpreted as an index institutional cohort and were compared descriptively with the supplied publications to identify shared patterns and important differences. Because the evidence base consisted mainly of case series, retrospective clinical data, and expert commentary, the review emphasizes qualitative synthesis and pragmatic clinical interpretation rather than pooled meta-analysis.

Results: Across the attached literature, carbide gun injuries consistently affected predominantly young males and commonly occurred when the user peeped into a gun after delayed or failed ignition, leading to combined thermal and alkali damage from acetylene combustion and calcium hydroxide residue. The five-patient central India case series reported a mean age of 16.2 years, universal monocular involvement, and presenting vision ranging from 6/36 to no light perception. The larger retrospective series included 28 eyes of 23 patients with a mean age of 28.48 years; severe injuries carried significantly poorer presenting and final visual acuity, more limbal stem cell deficiency, and occasional phthisis bulbi. Our institutional spreadsheet similarly showed that most patients were male, many were from urban or semi-urban settings, and several had corneal haze, epithelial defects, limbal ischemia, retained particulate matter, or need for amniotic membrane grafting. Taken together, the evidence indicates that carbide gun trauma is not a trivial firecracker

substitute but an evolving public health problem capable of causing long-term ocular surface failure and visual disability.

Conclusion: The cumulative evidence supports classifying carbide gun-induced ocular trauma as an emerging, preventable cause of severe visual morbidity, particularly in resource-limited Indian settings where cheap devices, easy availability of calcium carbide, and social media dissemination promote unsafe use. The institutional dataset mirrors the broader literature by demonstrating anterior segment burns, foreign body deposition, limbal ischemia, and variable but often guarded visual recovery after intensive treatment. Early ocular irrigation, debris removal, anti-inflammatory therapy, and timely surface-restoring surgery are central to management, yet prevention remains the most effective strategy because advanced burns frequently leave permanent scarring or limbal stem cell deficiency despite care.

Keywords: Calcium carbide; Carbide gun; Ocular trauma; Thermochemical injury; Visual morbidity.

INTRODUCTION

Carbide gun-induced ocular trauma has recently come into focus as an avoidable but potentially blinding form of eye injury in India. Although the device was originally used in agricultural settings to scare birds or animals away from crops, recent reports show that it is increasingly being repurposed for recreation and as a low-cost substitute for firecrackers. This shift is clinically important because the resulting injuries combine the destructive properties of thermal burns, alkali injury, mechanical blast, and particulate contamination.^[1-3] The underlying chemistry explains the severity of tissue damage. Calcium carbide reacts with water to generate acetylene gas and calcium hydroxide, and ignition of the accumulated acetylene produces an explosive burst with intense heat. The eye is then exposed not only to a pressure wave and flame but also to alkaline residue, most notably calcium hydroxide, which can penetrate the cornea and damage limbal stem cells. This thermochemical combination distinguishes carbide gun injury from simple thermal trauma and helps explain why even apparently localized exposure may progress to limbal ischemia, persistent epithelial defects, corneal scarring, and chronic ocular surface failure.^[2-3]

The available Indian literature suggests that the epidemiology of this injury pattern is changing. Earlier reports linked calcium carbide ocular burns to occupational exposure, such as fruit ripening work or agricultural use of scare guns. More recent studies, however, describe recreational injuries in children, adolescents, and young men who either purchased low-cost guns from informal vendors or assembled them using widely accessible online. The public health relevance is amplified by the fact that these patients belong largely to economically productive or school-going age groups and may require prolonged treatment, repeat surgery, and visual rehabilitation.^[2-4]

This review therefore aims to synthesize the available evidence contained in the attached materials and compare the institutional findings with

the published studies. The focus is on mechanism, demographic profile, clinical manifestations, management, outcomes, and prevention, with special attention to why this pattern should be regarded as an emerging cause of preventable visual morbidity in India.

MATERIALS AND METHODS

This review was designed as a systematic-style evidence synthesis of all documents provided in the attachment set, with the explicit objective of comparing the attached institutional findings with the supplied published studies.

Data sources (1) a public health editorial discussing ocular injuries from carbide guns and their relationship to social media dissemination; (2) an ophthalmic perspective article summarizing pathophysiology, clinical features, and management; (3) a retrospective analysis of sequelae in 28 eyes of 23 patients with calcium carbide-related thermochemical injury; (4) a case series of five children and young adults from central India; and (5) an Excel spreadsheet containing institutional clinical data on additional carbide gun injury patients.

Eligibility framework for inclusion in the synthesis, sources had to contain primary or clinically interpretable information related to ocular injury caused by calcium carbide or carbide guns. Opinion-oriented or editorial material was retained if it contributed clinically relevant descriptions of pathophysiology, burden, or prevention strategies. No language restriction was necessary because all supplied documents were in English.

Variables extracted The following variables were extracted when available: age, sex, place or context of injury, mode of exposure, laterality, time to presentation, presenting visual acuity, anterior and posterior segment findings, burn severity, acute management, need for surgery, follow-up, chronic sequelae, and final visual outcome. For the spreadsheet cohort, descriptive interpretation focused on age distribution, sex, rural/urban

background, time to presentation, laterality, ocular findings, and immediate management decisions.

Synthesis strategy Given the heterogeneity of study designs and the descriptive nature of most sources, formal meta-analysis was neither possible nor appropriate. Instead, a structured narrative synthesis was undertaken, centering on recurrent themes

across studies and then mapping the attached institutional cases against those themes. The main comparative domains were demographic profile, mechanism of injury, patterns of ocular involvement, treatment intensity, and visual prognosis.

RESULTS

Table 1: Comparison of demographic and exposure characteristics

| Parameter | Central India series | Retrospective analysis | Institutional spreadsheet | Comparative interpretation |
|-----------------------|---|---|---|---|
| Age profile | Mean 16.2 years, children/young adults | Mean 28.48 years, young adults | Ages range from 10 to 54 years, with clustering in teens and young adults | The institutional data bridge the pediatric and adult spectrum described in published studies |
| Sex | All male | Predominantly male | Mostly male, with one female bystander noted | Young male predominance is highly consistent across all datasets |
| Setting | Recreational use, local vendors, home-made device | Agricultural exposure common; some recreational use | Mostly active users, one bystander; both urban and rural cases | The present cohort resembles the newer mixed-use pattern rather than purely occupational exposure |
| Laterality | Universal monocular injury | Severe burns often unilateral; some bilateral milder injuries | Mostly unilateral, one bilateral/bystander entry | Unilateral direct-viewing injuries dominate the severe phenotype |
| Delay to presentation | Within 24 hours | Median 1 day | Many presented within hours to 1 day, though some were delayed several days | Early presentation is common, but delayed cases still occur and may worsen prognosis |

Table 2: Comparison of ocular findings and management

| Domain | Central India series | Retrospective analysis | Institutional spreadsheet |
|--------------------------|--|--|--|
| Typical surface findings | Conjunctival congestion, dense corneal haze, epithelial defects, limbal ischemia, periocular burns | Corneal scar, foreign body deposits, LSCD, persistent sequelae after acute burns | Congestion, subconjunctival hemorrhage, corneal haze, epithelial defects, limbal ischemia, carbide particles |
| Severe injury markers | 270-360 degrees limbal ischemia, dense haze, PL vision, AMT requirement | Grade IV-VI burns, poorer presenting and final BCVA, phthisis in 2 eyes | Total epithelial defect, dense haze, limbal ischemia, media obscuration, AMG advised/performed |
| Initial care | Saline irrigation, topical steroids, antibiotics, lubricants, cycloplegics, oral vitamin C | Irrigation, debris removal, topical medications, oral vitamin C in severe group | Eye wash, foreign body removal, topical drops, oral/systemic medication, intravenous methylprednisolone |
| Surgery | AMT with symblepharon ring in severe burns | AMT, tarsorrhaphy, globe repair, VR surgery, later CAG/SLET/PK in selected cases | AMG advised or performed in several severe presentations |

Table 3: Outcome comparison and implications

| Outcome feature | Central India series | Retrospective analysis | Institutional spreadsheet | Implication |
|--------------------------|---|---|---|---|
| Final visual recovery | Mild-moderate cases improved to 6/12-6/18 or 6/24; severe cases remained poor | Group A median final BCVA 20/32 versus 20/200 in Group B | Final outcomes are incompletely standardized, but severe eyes required aggressive therapy | Severity at presentation strongly shapes prognosis |
| Long-term surface damage | Persistent corneal opacity in severe cases | LSCD in 6/16 Group A and 9/12 Group B eyes; phthisis in 2 severe eyes | Limbal ischemia and epithelial defects suggest future LSCD risk in severe local cases | Carbide trauma is a chronic ocular surface disease risk, not just an acute burn |
| Public health concern | Unsafe sale, parental unawareness, online construction advice | Need for awareness and advocacy emphasized | Mixed urban-rural occurrence indicates broad community penetration | Prevention must target supply, supervision, and public messaging simultaneously |

The attached literature reveals a remarkably coherent injury pattern despite differences in design and sample size. Carbide gun trauma predominantly affects males in the pediatric to young adult age range and typically follows ignition failure, delayed

explosion, or inspection of the device from the muzzle side. This pattern is reflected in both formal publications and the institutional spreadsheet, supporting the view that the syndrome is reproducible and clinically distinctive.

Demographic profile The central India case series documented five injured males with a mean age of 16.2 years. The retrospective LVPEI study reported a mean age of 28.48 years, again with clear male predominance. The institutional spreadsheet broadens this age range from 10 to 54 years, but the majority remain adolescents or young adults, with only one female bystander entry. Taken together, these findings indicate that the burden falls mainly on active users who are often within school-going or working-age groups.

Mechanism Peering into the barrel or opening after failed or delayed firing. The blast exposes the eye to flame, pressure, residual calcium hydroxide, and sometimes particulate debris, producing a combined thermochemical injury

Ocular phenotype The dominant ocular findings include conjunctival congestion, subconjunctival hemorrhage, corneal epithelial defects, stromal haze or opacity, limbal ischemia, periocular burns, and retained white particulate material. Severe injury is characterized by extensive limbal ischemia, poor presenting vision, and total or near-total epithelial loss. In the retrospective study, severe burns were associated with significantly worse baseline and final acuity as well as higher rates of limbal stem cell deficiency and phthisis. The spreadsheet cases replicate this spectrum, including limbal ischemia, total epithelial defect, dense haze, corneal and conjunctival foreign bodies, and obscured media in severe eyes.

Visual and structural outcomes Visual recovery depended strongly on initial severity. In the five-patient series, milder cases improved to 6/12, 6/18, or 6/24, whereas the two most severe cases remained at finger counting close to face or 3/60 despite treatment. In the larger retrospective series, the median final best-corrected visual acuity was 20/32 in Grades I-III and 20/200 in Grades IV-VI, confirming a substantial gap in recovery between lower- and higher-grade burns. Chronic sequelae included corneal scarring, limbal stem cell deficiency, symblepharon, and phthisis bulbi in the most severe eyes. These data support the conclusion that even when patients present early and receive appropriate therapy, severe carbide injury frequently results in lasting ocular surface morbidity.

Statistical Analysis

The retrospective analysis provided the strongest inferential data and demonstrated statistically significant differences between lower-grade and higher-grade burns. Specifically, the median presenting BCVA was significantly worse in the severe-burn group than in the mild-to-moderate group, with a reported P value of 0.002. Final median BCVA also remained significantly worse in the severe group, with a P value of 0.02, despite treatment. By contrast, the duration of follow-up did not differ significantly between groups, with a reported P value of 0.24.

DISCUSSION

The attached studies collectively indicate that carbide gun ocular trauma should be understood as a modern variant of preventable blast-related ocular burn, shaped by local cultural practices, informal manufacturing, and digital dissemination. The injury is not merely a thermal burn and not merely a chemical burn; instead, it is a compounded thermochemical event often intensified by close-range viewing and mechanical debris. This distinction matters clinically because management must address pH neutralization, retained particles, epithelial regeneration, inflammation control, and long-term ocular surface reconstruction.^[1-3]

A major contribution of the supplied evidence is the demonstration that recreational use now sits alongside agricultural use as a major exposure context. The central India series specifically documented purchases from street vendors or local shops and even home construction with online guidance. The editorial likewise frames social media as a catalyst for re-emergence of these devices among children and adolescents. The institutional spreadsheet, which includes several urban cases, further suggests that this is no longer a niche rural occupational hazard.^[2]

The comparison between the central India series and the retrospective multicenter study is especially informative. The former highlights the adolescent recreational profile and the sociobehavioral factors behind exposure, while the latter maps the full trajectory from acute burn to chronic sequelae. Together, they show that the same device can generate both immediate tissue destruction and delayed ocular surface failure requiring staged surgical rehabilitation. The institutional cases are concordant with this continuum, as several demonstrate severe anterior segment injury at presentation and escalation to amniotic membrane grafting or systemic therapy. Another important lesson is that early presentation does not guarantee favorable vision. In both major clinical studies, many patients reached medical care within a day, and irrigation plus standard acute therapy were instituted promptly. Nevertheless, severe limbal ischemia and dense corneal haze often translated into poor visual endpoints. This underscores the biological aggressiveness of alkali-associated limbal injury and supports aggressive early surface preservation strategies.^[2-4]

The public health implications are substantial. Victims are frequently schoolchildren, adolescents, or young working men, meaning the functional cost of blindness or ocular surface disability is magnified socially and economically. Reconstructive procedures such as SLET, conjunctival autografting, or penetrating keratoplasty may not be readily accessible in all regions and impose additional costs on families and health systems. Because the injury is largely avoidable, prevention should be prioritized

over reliance on tertiary care rescue. The present institutional spreadsheet strengthens the translational relevance of the literature. Its cases resemble the published experience closely enough to suggest that the observed syndrome is not isolated to one center or one festival season. The recurring signs of limbal ischemia, epithelial defect, carbide particle deposition, dense haze, and need for ocular surface-focused treatment indicate a shared clinical template that ophthalmologists should recognize rapidly.^[3-5]

The five-patient series from central India offers a clear picture of the emerging pediatric and adolescent burden. In that study, all injured patients were male, the mean age was 16.2 years, all injuries were monocular, and the precipitating event was typically accidental ignition while looking into a malfunctioning barrel. The authors highlighted several enabling factors, including poor parental awareness, easy availability, lack of warning labels, and the appeal of a cheap firecracker alternative. Parents frequently recommended sale restrictions or bans after the incident, reinforcing the preventable nature of the hazard. The larger retrospective study from LV Prasad Eye Institute further shows that these injuries are not only acute emergencies but also sources of chronic morbidity. Among 28 eyes of 23 patients included after adequate follow-up, more severe burns were associated with significantly worse presenting and final visual acuity. Limbal stem cell deficiency developed in a substantial fraction of eyes, especially severe burns, and two eyes progressed to phthisis bulbi. The need for amniotic membrane transplantation in most moderate-to-severe cases indicates that the injury often extends beyond a superficial epithelial insult and may compromise the full ocular surface ecosystem.^[2,5,6]

The attached institutional spreadsheet supports these published observations and adds practical local context. The dataset includes multiple young male patients, chiefly with closed-globe anterior segment injury patterns characterized by conjunctival congestion, subconjunctival hemorrhage, corneal haze, limbal ischemia, epithelial defects, and embedded carbide particles. Several cases required operating room foreign body removal, systemic therapy, or amniotic membrane grafting, again underscoring that these are not self-limited minor injuries. From a research perspective, carbide gun ocular trauma remains under-reported relative to its apparent clinical seriousness. Most available evidence comes from case reports, case series, retrospective studies, and expert commentary rather than prospective multicenter registries. Yet the consistency of the clinical pattern across different sources strongly suggests a recognizable syndrome with major preventive implications.^[7]

At the same time, this review has limitations. The available evidence remains observational, numbers are modest, and some attachments represent commentary rather than primary research. The

spreadsheet data are not fully normalized for statistical pooling, and long-term outcomes are incomplete for several local cases. Even so, when independent sources repeatedly describe the same mechanism, phenotype, and consequence pattern, the signal is strong enough to justify both clinical vigilance and preventive action. The pathophysiology of injury is particularly destructive because it combines thermal burn, alkali chemical burn, blast effect, and particulate deposition. This explains why a seemingly small device can produce severe limbal ischemia, dense corneal haze, total epithelial loss, and long-term ocular surface instability. The strongest clinical message from the supplied evidence is that severity at presentation determines prognosis: mild-to-moderate cases may recover useful vision, whereas severe burns often culminate in corneal scarring, limbal stem cell deficiency, repeated surgery, and poor final acuity.^[8-12]

Comparison of the present institutional findings with the provided studies shows substantial concordance. The same recurring markers of danger are visible across datasets: male predominance, unilateral close-range exposure, corneal particulate matter, epithelial defects, limbal ischemia, obscured media in severe eyes, and need for urgent eye wash plus escalated therapy. This convergence enhances confidence that carbide gun trauma represents a reproducible clinicopathologic entity rather than a scattered anecdotal phenomenon. In management terms, prompt and thorough irrigation remains the cornerstone of acute care, followed by meticulous removal of deposits, intensive topical therapy, lubrication, cycloplegia, and adjunctive systemic measures in more severe burns. Amniotic membrane transplantation appears central in the acute management of moderate-to-severe ocular surface injury, while later-stage procedures such as conjunctival autografting and simple limbal epithelial transplantation may be required when limbal stem cell deficiency or cicatricial sequelae develop. Despite these options, even optimal care cannot reliably reverse the consequences of the most severe burns.^[13-17]

CONCLUSION

Carbide gun-induced ocular trauma is an emerging and highly preventable cause of visual morbidity that deserves recognition as a distinct public health and ophthalmic emergency. The attached literature consistently shows that the affected population is dominated by children, adolescents, and young adult males, with injuries occurring in both agricultural and recreational contexts. The attached institutional spreadsheet confirms that this profile is not theoretical or distant but locally relevant, with similar injuries appearing in urban as well as rural patients. For that reason, the principal solution is prevention. Effective countermeasures should

therefore include restriction of unlicensed sale of carbide guns and calcium carbide for recreational use, age-based access control, public awareness campaigns before festive seasons, school and parent education, and stronger moderation of online content that glamorizes device construction or use. From an academic standpoint, the topic warrants more robust prospective study.^[18-20]

REFERENCES

1. Tandon R, Sahay P, Goyal D. Ocular injuries from carbide guns: Public health risk in the social media era.
2. Mohan S. Carbide Gun Injury: An Ophthalmic Perspective.
3. Arora A, Shanbhag SS, Mishra DK, Malepati N, Das S, Priyadarshini SR, et al. Sequelae of carbide-related thermochemical injury: A retrospective analysis.
4. Sabde YD, Trushna T, Bhartiya P, Tiwari RR. Vision-threatening ocular injury secondary to calcium carbide gun use in Indian children and young adults: A case series.
5. Bandyopadhyay S, Saha M, Biswas S, Ranjan A, Naskar AK, Bandyopadhyay L. Calcium carbide related ocular burn injuries during mango ripening season of West Bengal, eastern India.
6. Sanjay S, Gupta A, Uday S. Scare air gun-induced ocular trauma in an agriculture farm in India.
7. Sharma N, Kaur M, Agarwal T, Sangwan VS, Vajpayee RB. Treatment of acute ocular chemical burns.
8. Dua HS, King AJ, Joseph A. A new classification of ocular surface burns.
9. Roper-Hall MJ. Thermal and chemical burns.
10. Veldman PB, Greiner MA, Cortina MS, Kuo AN, Li JY, Miller DD, et al. Efficacy of amniotic membrane grafting for the treatment of chemical and thermal ocular surface injuries.
11. Clare G, Bunce C, Tuft S. Amniotic membrane transplantation for acute ocular burns.
12. Shanbhag SS, Patel CN, Goyal R, Donthineni PR, Singh V, Basu S. Simple limbal epithelial transplantation (SLET): Review of indications, surgical technique, mechanism, outcomes, limitations, and impact.
13. Basu S, Sureka SP, Shanbhag SS, Kethiri AR, Singh V, Sangwan VS. Simple limbal epithelial transplantation: Long-term clinical outcomes in unilateral chronic ocular surface burns.
14. Agarwal T, Vajpayee RB, Sharma N, Tandon R. Severe ocular injury resulting from chuna packets.
15. John D, Philip SS, Mittal R, John SS, Paul P. Spectrum of ocular firework injuries in children: A 5-year retrospective study during a festive season in Southern India.
16. Herr RD, White GL Jr, Bernhisel K, Mamalis N, Swanson E. Clinical comparison of ocular irrigation fluids following chemical injury.
17. Rihawi S, Frenzt M, Becker J, Reim M, Schrage NF. The consequences of delayed intervention when treating chemical eye burns.
18. Westkemper H, Figueiredo FC, Siah WF, Wagner N, Steuhl KP, Meller D. Clinical outcomes of amniotic membrane transplantation in the management of acute ocular chemical injury.
19. Shanbhag SS, Chanda S, Donthineni PR, Basu S. Surgical management of unilateral partial limbal stem cell deficiency: Conjunctival autografts versus simple limbal epithelial transplantation.
20. World Health Organization and UNICEF. World report on child injury prevention.