January - June 2025 Volume 34 Issue 1 PRINT ISSN: 2277-1867
ONLINE ISSN: 2277-8853

JOURNAL OF FORENSIC MEDICINE SCIENCE AND LAW

Official Publication of Medicolegal Association of Maharashtra

MULTISPECIALITY, MULTIDISCIPLINARY, NATIONAL
PEER REVIEWED, OPEN ACCESS, MLAM (SOCIETY) JOURNAL
Indexed with Scopus (Elsevier)

Editorial Office Address
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(Official Publication of Medicolegal Association of Maharashtra)
Email.id: mlameditor@gmail.com

PRINT ISSN: 2277-1867
ONLINE ISSN: 2277-8853

Case Report

Carbon Deposition in Alveolar Structures: A Marker of Ante-Mortem Smoke Inhalation

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Article Info

Received on: 06.03.2025
Accepted on: 01.06.2025

Key words
Soot Deposition, Smoke Inhalation, Antemortem Burns, Fire-Related Deaths.

Abstract

In **fire related deaths**, especially with no history or injuries, it is not always clear whether death was caused by burns, smoke, or both. In such cases, studying the lungs under a microscope becomes very important, especially when obvious burn injuries are absent. Smoke inhalation shows a recognizable histopathological finding such as congestion of blood vessels, swelling of capillaries, bleeding into the air sacs, and damage to the delicate alveolar walls. The presence of soot particles in the trachea, along with these microscopic changes, strongly suggests that the person was alive and breathing during the fire. However, **carbon particles** seen inside the alveoli but not deeper in lung tissue indicate that they were inhaled rather than passively deposited after death. More convincing evidence is the presence of macrophages actively engulfing carbon particles—proof of a living response to smoke inhalation. This study highlights the value of routinely examining the lungs histologically in fire-related deaths. Doing so helps forensic experts distinguish between injuries sustained before and after death, while also deeply understanding the pathogenesis of smoke inhalation injury in causing death.

1. Introduction

The percentage of burn patients who experience inhalation injury—which consistently stands at around 15–20% of hospital admissions internationally—represents an important aspect of burn care over several decades, as demonstrated by comprehensive systematic reviews and research from multiple countries.[1] Autopsy examinations play a pivotal role in establishing the cause of sudden or unexplained deaths, as well as confirming uncertain clinical diagnoses. Over 96% of fire-related deaths worldwide occur in developing and underdeveloped countries, with the South-East Asia region bearing a disproportionately high share of the global burden. These elevated mortality rates are attributed to factors such as poor infrastructure, inadequate fire safety education, overcrowded living conditions, and insufficient emergency medical services.[2] Accidental burns commonly occur both in domestic and occupational settings. Within households,

How to cite this article: Sabale P, Sankholkar C, Kavishwar V, Kumar PP. Carbon Deposition in Alveolar Structures: A Marker of Ante-Mortem Smoke Inhalation. J Forensic Med Sci Law. 2025;34(1):67-71.
doi: 10.59988/jfmsl.vol.34issue1.14

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frequent causes include electrical short circuits, clothes catching fire whilst cooking, leakage of cooking gas, and accidents involving lamps or candles. Vulnerable groups include children, elderly, and individuals under the influence of alcohol or drugs. In occupational environments, ranging from small-scale industries to heavy industrial operations, burns remain prevalent and contribute significantly to morbidity and mortality.

One of the major forensic challenges in burn-related deaths is the distinction between antemortem and postmortem burns. The most reliable indicators of antemortem burn and smoke inhalation injuries include the presence of soot particles in the trachea or lower respiratory tract and elevated concentrations of carboxyhaemoglobin in the blood. However, the absence of carboxyhaemoglobin does not exclude antemortem burn injuries, as carbon monoxide generation may be minimal in open-air fires or if the person died rapidly before inhaling large quantities.

Owing to their extremely small size, soot particles can passively deposit within the upper respiratory tract. However, the deposition of soot particles within the trachea and extending into the lower respiratory tract, including terminal bronchioles, indicates active respiration during the fire. This vital reaction usually reflects increased breathing secondary to fear, or smoke-induced airway compromise, confirming that inhalation occurred before death which is usually considered as a telltale sign of antemortem inhalation injury,

Nevertheless, the absence of soot particles in the trachea or bronchi does not exclude antemortem exposure. Several factors may explain such cases, including burn incidents in open spaces where soot is rapidly dispersed by environmental airflow, or cases where the victim survived long enough to receive oxygen therapy and resuscitative interventions that can remove or redistribute carbon particles within the airway. These intricacies highlight the importance of integrating both gross and microscopic findings along with biochemical evidence to accurately establish the role of inhalational injury in burn-related deaths.

2. Case summary

An 82-year-old female was brought to the hospital with a history of breathlessness and loss of consciousness at home due to inhalation of smoke, which developed due to a short circuit in their building. She died within 15 minutes of admission while she was on treatment. She had no history of smoking or any occupational exposure that would significantly contribute to the histopathological findings mentioned below.

3. Pathological findings

On examination there were no external injuries. Soot particles with mucosal secretions were seen in the tracheal lumen on gross examination (**Figure 1, Figure 2**). Histopathology examination with haematoxylin and eosin stain revealed the presence of carbon particles in alveolar walls (**Figure 3, Figure 4**) and spaces along with alveolar collapse and oedema.

Figure 1: Soot in Tracheal lumen.

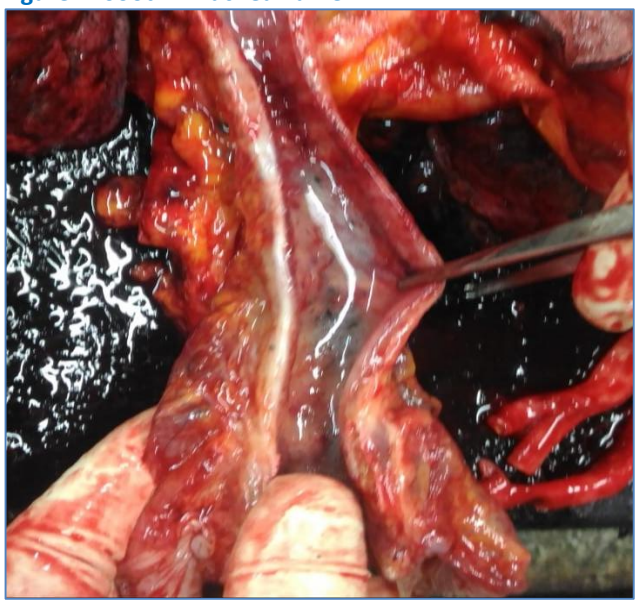
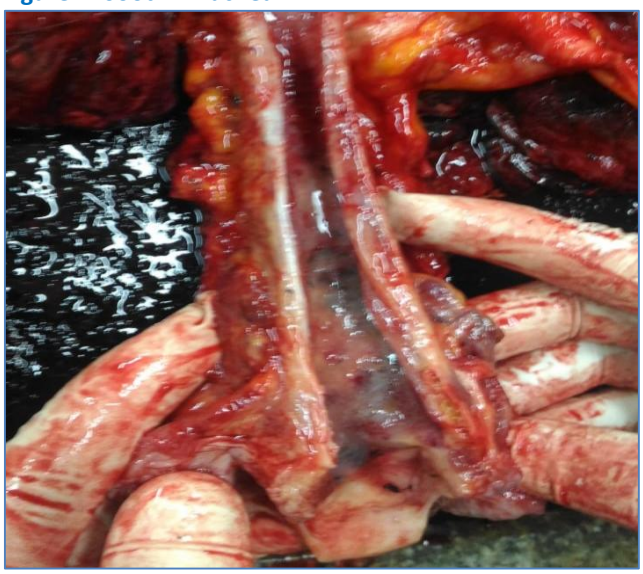


Figure 2: Soot in Trachea.



4. Discussion

In this study, histopathological examination revealed the presence of carbon particles along the alveolar walls in cases of smoke inhalation injury.

Several mechanisms have been proposed to explain the origin of these particles within the lung:

1. Deposition of exogenous soot directly inhaled into the alveoli.
2. Release of endogenous elemental carbon during altered carbon dioxide metabolism, and
3. Accumulation of endogenous blood-derived pigment.[3]

Figure 3: Prominent deposits along alveolar wall (100X).

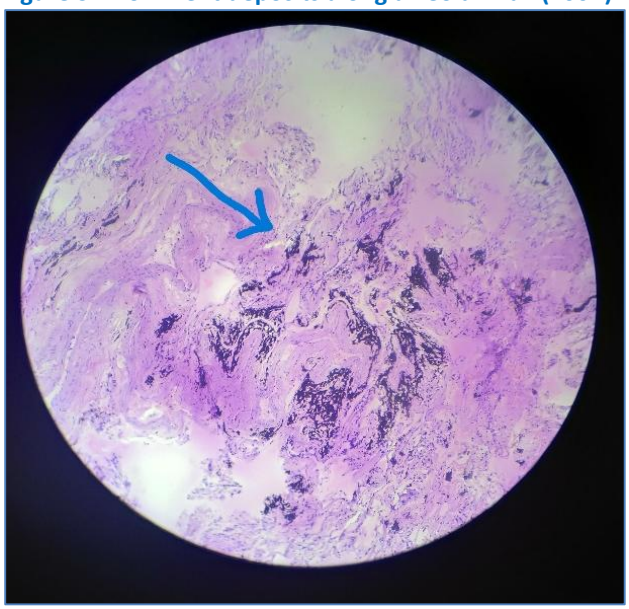
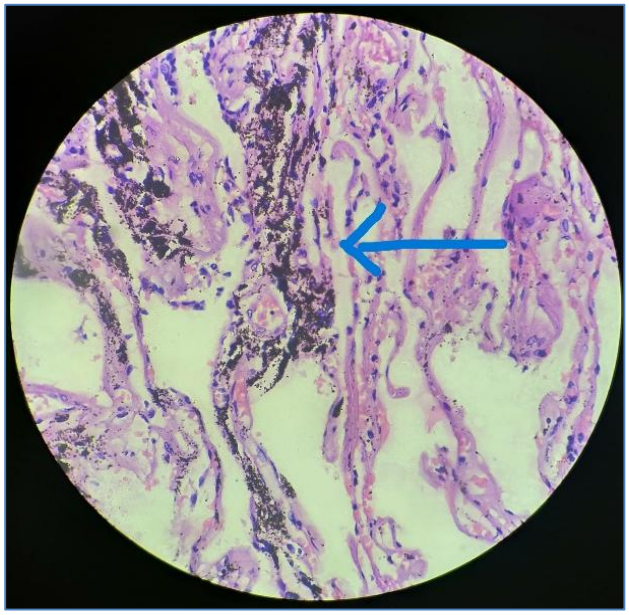


Figure 4: Deposits in alveolar wall (450X).



The endogenous elemental carbon usually is deposited within the deeper layers of the lung parenchyma unlike this case where carbon particles were noted along the alveolar walls and endogenous blood derived pigments can be confirmed with Perl's stain which was not appreciated in this case. Pinkerton et al. demonstrated that carbonaceous particulate matter can be identified in subpleural septa and lymphatics even in nonsmokers, suggesting an important contribution of background environmental exposure.[4]

Similarly, Podovan et al. emphasized that smoking-related carbon deposition may be secondary to ambient air particulate exposure rather than smoking alone.[5] A notable forensic report by Senarath et al. described the presence of soot beyond secondary bronchioles, accompanied by pulmonary congestion and haemorrhage, in a family who died in a house fire. These findings strongly supported soot deposition as a vital sign of ante-mortem inhalation.[6] Rahimi et al. further confirmed the systemic distribution of black carbon particles by identifying them in vascular spaces of multiple organs. These particles were distinguishable from hemosiderin by negative Perl's staining, highlighting their origin as inhaled particulate matter rather than endogenous pigment.[7]

Characteristic histopathological patterns have also been documented in burn-related smoke inhalation cases as "fire lungs" present with bronchiolar dilation and alveolar haemorrhage, while "suffocation lungs" typically show alveolar collapse and vascular congestion. Megahed et al. demonstrated a significantly higher mortality rate among patients with inhalation injury compared to those without, underscoring the clinical and prognostic significance of these findings.[8] Nemmar et al. showed that ultrafine particles are capable of translocating rapidly into systemic circulation, most likely through the alveolar-blood barrier rather than via phagocytosis, thereby contributing to extrapulmonary effects.[9] This concept is further supported by the detection of pneumoproteins in circulation, reflecting increased permeability of the alveolar-capillary barrier.[10] Ultrastructural studies have also documented phagocytosis of carbon particles by alveolar macrophages and associated epithelial injury, reinforcing inhaled soot as a clear marker of vitality.[11] Reviews of smoke inhalation pathology consistently describe progressive patterns of damage: epithelial necrosis, inflammatory cell infiltration, and increased vascular permeability, all of which highlight the destructive cascade initiated by inhaled smoke exposure.[12]

Postmortem studies combining imaging with histopathology corroborate these changes, revealing soot deposition and pulmonary oedema as hallmark features of ante-mortem smoke inhalation.[13]

Furthermore, Gupta et al. provide a detailed discussion of the etiopathogenesis, linking soot deposition with alveolar wall destruction, vascular congestion, and widespread tissue injury.[14] Taken together, our findings are consistent with these prior observations. The demonstration of carbon particles along alveolar walls and within phagocytic macrophages represents a reliable and definitive indicator of ante-mortem smoke inhalation. This not only aids in forensic interpretation of burn-related deaths but also deepens our understanding of the pathological processes underlying smoke-induced lung injury. Explosions and inhalational injuries at workplaces—both in large industries and informal sectors—are a major cause of death. In India, occupational safety and health standards are established by law and are enforced by agencies such as the Directorate General Factory Advice Service & Labour Institutes (DGFASLI) under the Government of India, along with inspectorates of factories and boilers at the state level.[15] Burn injuries are a significant public health problem in India, with millions affected annually and high mortality attributed to socio-economic factors, including poverty and limited access to specialized care. National programs have been instituted to improve prevention and management, recognizing the challenge posed by burns in the society.[16]

5. Conclusion

Surface soot in the trachea and bronchi and carbon deposits in alveolar walls or macrophages are crucial forensic markers of ante-mortem smoke inhalation. These findings help differentiate ante-mortem from postmortem burns and should be assessed alongside clinical context for accurate medicolegal diagnosis.

6. Recommendations

Further studies with larger samples are needed to consolidate soot and mucus-laden macrophages as definitive markers of life at the time of fire exposure.

Contributor ship of Author: All authors equally contributed.

Conflict of interest: None to declare.

Source of funding: None to declare.

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