Histopathological assessment of lung tissue under impact

Khyati Verma, Arul Selvi, Anoop Chawla, Sudipto Mukherjee

Abstract Blunt chest injuries are a common injury concern among civilian populations, commonly caused by road traffic accidents, falls, and crush injuries. These injuries often extend beyond the bony structures to involve critical underlying organs, such as the lungs, heart, and aorta. The severity of lung injuries is closely influenced by the mechanism and magnitude of impact, making it essential to understand these parameters for effective clinical management. This study investigates the effects of impact velocity on the lungs and explores the resultant microstructural damage. Using a modified electromechanical gun, goat lungs were subjected to controlled impacts at varying velocities (1–3 m/s). Histological analyses of the impacted lung tissue revealed that microstructural damage, such as collapsed alveoli (atelectasis), occurs regardless of the magnitude of the impact velocity.

Keywords Dynamic compression, lung injury, histology of lung tissue, road traffic accidents, histopathological analysis.

I. INTRODUCTION

The incidence of blunt chest injuries, particularly non-penetrating types, has risen significantly due to the increase in road traffic accidents. For instance, a review by Liman, *et al.* [1] of 1,490 patients over two years reported a mortality rate of 1% in cases of blunt chest trauma. While such trauma often remains confined to the bony structures of the chest, it can lead to serious pathophysiological alterations in the underlying soft tissues, such as the lungs, potentially resulting in fatal complications if not treated promptly [2]. Non-penetrating chest trauma frequently manifests as pulmonary contusion, characterised by interstitial and alveolar injury without visible lacerations. Diagnostic techniques such as computed tomography (CT) and chest radiography play a crucial role in identifying lung abnormalities caused by blunt impact. Among these, CT is regarded as the most accurate method for assessing pulmonary parenchyma and the pleural cavity in blunt trauma cases [3]. The mechanisms of lung injury caused by blunt trauma have been explored through various hypotheses. One study [4] proposed that sudden compression of the lungs causes bursting due to the inability of air to escape through normal airways, while another [5] suggested that laryngeal closure during compression increases air pressure, leading to lung rupture. Such injuries can result in complications such as atelectasis, interlobular haematoma, emphysema, lung torsion, and lung hernia [6].

Pulmonary contusion volume, measurable via admission CT scans, correlates with injury severity and complications like acute respiratory distress syndrome (ARDS), highlighting the importance of early interventions to reduce morbidity and mortality [7]. However, outcomes of blunt chest trauma are influenced by multiple factors, necessitating a multidisciplinary approach to treatment. Pulmonary contusion, prevalent in blunt thoracic trauma cases, lacks reliable diagnostic techniques, necessitating an integrated clinical and biomechanical approach to enhance understanding and treatment [8]. Bridging the clinical and mechanical perspectives of blunt lung injury is essential for understanding its mechanisms and improving treatment strategies. This study addresses these gaps by integrating clinical insights with mechanical testing to explore the effects of dynamic compression on the lungs. It focuses on investigating microstructural damage resulting from varying impact speeds, providing a foundation for improved identification, treatment, and mitigation of blunt lung trauma.

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II. METHODS

Experimental Testing

Dynamic impact tests were conducted on isolated goat lungs using a custom-designed electromechanical impact gun to study the effects of varying impact velocities (1–3 m/s) [9]. Impact velocities were controlled by adjusting the spring compression mechanism. The lungs were positioned on a circular plate mounted on a load cell (ADI ARTECH, S-type, 100 kg), with the assembly's height being adjustable via a screw jack, as illustrated in Fig. 1. High-speed cameras (20,000 frames/s) recorded the impact, enabling precise calculation of impact velocity and displacement through marker tracking on the impactor. An electronic trigger synchronised the force-displacement data, marking the event onset.

Post-testing, samples were fixed in a 10% formalin solution for 48 hours to preserve tissue integrity. To ensure thorough penetration and prevent autolysis, incisions were made on the samples, avoiding the impacted regions. Before and after fixation, the samples' dimensions and shape were documented. Gross examinations identified superficial damage, including lacerations and contusions, as discussed in subsequent sections. Histopathological analysis involved tissue sampling from the impacted area, adjacent zones, and distal regions [10].

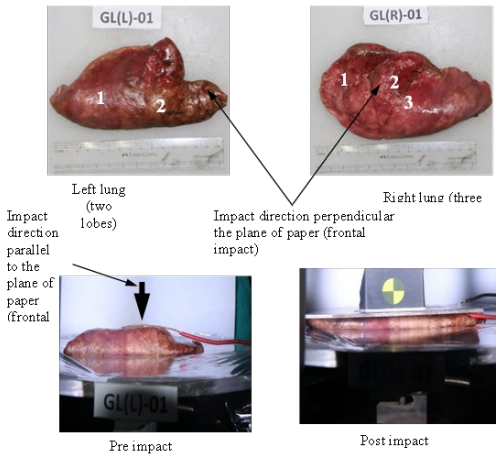

Fig. 1. Whole organ arrangement and sample placement for dynamic compression test [10].

Gross Examination

Superficial damage was assessed through visual inspection, while deeper tissue damage was evaluated histologically. The effects of the impact on areas adjacent to the point of contact were also analysed. External signs such as blood vessel damage or alveolar rupture were absent due to the post-mortem condition of the samples, which lacked circulating blood. Penetrative injuries, such as lacerations, were not observed because the isolated organs were examined without the surrounding bony structures. Tissue sections from the impacted, adjacent, and unaffected regions were prepared for detailed microstructural analysis.

Slide Preparation for Microscopy

Three rectangular tissue sections, representing the impacted, adjacent, and unaffected areas, were prepared for histological analysis. The tissues were initially fixed in 10% formalin to preserve their structure. After fixation, dehydration was carried out by immersing the tissues in graded alcohol solutions.

Xylene was subsequently used as a clearing agent to replace the dehydrating fluid. The cleared tissues were embedded in molten paraffin wax to provide structural support for sectioning. Thin slices, approximately 2 microns thick, were cut into ribbons using a microtome and transferred to warm water before being mounted onto glass slides. The slides were then stained with haematoxylin and eosin to enhance cellular structures, making them suitable for microscopic examination (Fig. 2).

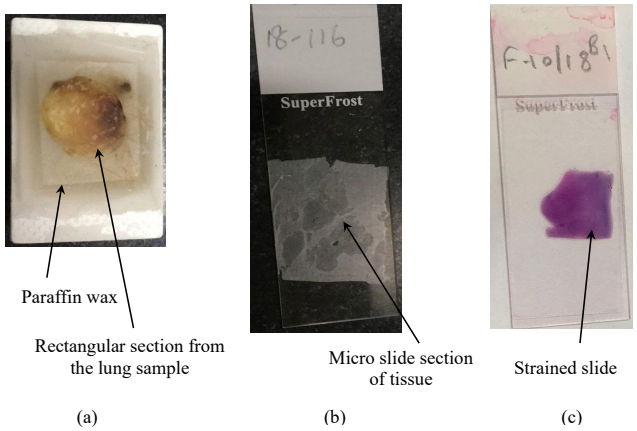

Fig. 2. (a) Sample embedded in paraffin wax; (b) micro slide section; (c) staining of the micro slide section.

III. INITIAL FINDINGS

Superficial damages were assessed through visual inspection, while histological analysis was used to evaluate deeper injuries. Due to the post-mortem nature of the samples, which lacked blood, surface damage, such as blood vessel rupture and superficial alveolar bursting, was not observed. Lacerations were also absent as the test involved isolated organs without bony structures. For detailed analysis, tissue sections were obtained from the impacted, adjacent, and unaffected (normal) areas. Histological findings revealed collapsed alveoli regardless of impact velocity, coalesced emphysematous spaces, and focal clusters of chronic inflammatory cells in some samples (Fig. 3).

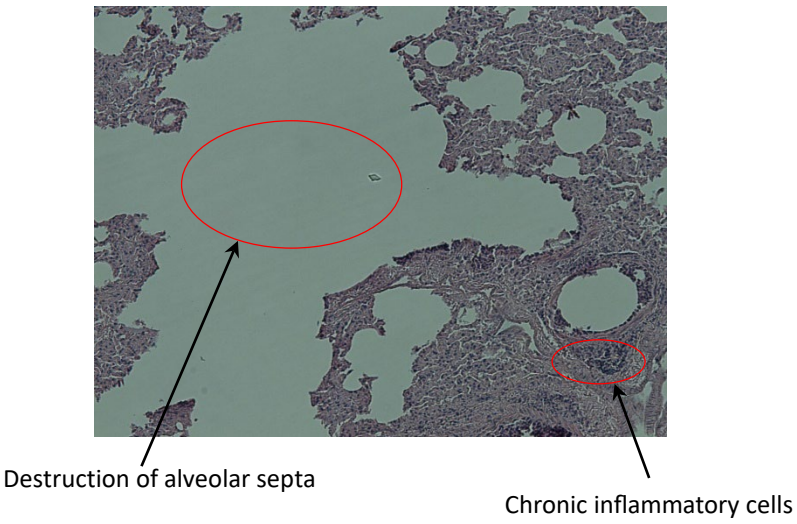

Fig. 3. Irregular alveolar spaces and sites of chronic inflammatory cells in the lung.

IV. DISCUSSION

Histological analysis of lung tissue provides critical insights into the microstructural damage caused by external forces and pathological conditions. Healthy lung tissue, characterised by thin-walled alveoli lined with squamous epithelium and interconnected by connective tissue, demonstrates remarkable adaptability under normal conditions. However, excessive deformation due to impact results in significant structural alterations. The observed atelectasis (collapsed alveoli) and emphysema (irregular air spaces) are direct consequences of increased internal alveolar pressure, leading to tissue compromise. These changes, if untreated, could escalate into life-threatening conditions, emphasising the importance of timely intervention. Chronic inflammatory cells and fibrosis observed in some samples did not significantly influence the impact-induced damage, indicating that these pathological conditions may have minimal interaction with mechanical deformation processes. This finding aligns with the understanding that mechanical stresses and biological factors independently contribute to lung tissue integrity under +different contexts. The results underline the critical need for a multidisciplinary approach that combines histological investigation with advanced computational modeling, such as finite element (FE) simulations. Establishing correlations between microstructural damage and stress-strain data can refine predictive models of lung injury, providing a foundation for improved diagnostic and therapeutic strategies.

V. CONCLUSION

This study highlights the significant microstructural changes in lung tissue resulting from external impact, specifically atelectasis and emphysema. The absence of superficial injuries in the samples requires the necessity of detailed histological evaluation to uncover underlying damage. Findings suggest that while chronic pathological conditions such as fibrosis and inflammation do not aggravate mechanical injury outcomes, they do underscore the complex interplay between structural and biological factors in lung health. Future studies with a larger sample size will be essential to establish a robust correlation between histological findings and mechanical stress data derived from FE simulations. This integration could enhance clinical management by enabling early detection of severe injuries and guiding personalised therapeutic interventions.

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