

Hepatic Effects of Chronic Occupational Cement Dust Exposure: Enzyme Profile Analysis

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ABSTRACT

Background

Occupational cement dust exposure poses significant health risks, with recent evidence suggesting potential impacts on liver function, particularly in developing countries with expanding cement industries.

Objectives

This study aimed to evaluate the effect of chronic occupational cement dust exposure on liver function among male adults working in a cement factory in Bangladesh, assessing serum liver enzyme levels as biomarkers of hepatic stress.

Methods

A cross-sectional study was conducted from September 2017 to August 2018 at Dhaka Medical College, Dhaka, and Ninety-two healthy males aged 20-50 years were enrolled, including 46 workers exposed to cement dust for at least 2 years working in a Munshiganj-based cement mill in Bangladesh and 46 matched controls with no occupational exposure. Liver enzymes, Serum Glutamic Pyruvic Transaminase (SGPT) and Serum Glutamic-Oxaloacetic Transaminase (SGOT), and alkaline phosphatase (ALP) were assessed using automated analyzers. Statistical analyses included the Mann-Whitney U test, the Pearson correlation coefficient test, and logistic regression.

Result

Mean liver enzyme levels (SGPT and SGOT) were significantly reduced while serum ALP increased dramatically in exposed workers compared to controls, with a p-value of <0.001. Though all values remained within the normal clinical range, the significant alterations in liver enzymes upon cement dust exposure may indicate early subclinical hepatic stress. The liver enzyme alterations likely reflect chronic, low-level inflammatory and oxidative challenges, with differences attributed to workplace exposure intensity and duration.

Conclusions

Chronic occupational exposure to cement dust may lead to measurable subclinical alterations in liver enzymes, suggesting early hepatic adaptation or stress despite an absence of overt disease. Regular monitoring and improved workplace safety measures are recommended to mitigate long-term risks.

Keywords

Cement dust exposure, Liver function tests, Serum Glutamic-Oxaloacetic Transaminase (SGOT), Serum Glutamic Pyruvic Transaminase (SGPT), alkaline phosphatase (ALP), Occupational health, Subclinical liver injury, Bangladesh workers.

INTRODUCTION

Occupational exposure to cement dust is recognized worldwide as a significant health challenge, particularly in developing countries where the cement industry continues to expand rapidly ¹. Cement is essential for the modern construction sector. Still, its manufacture and handling release substantial amounts of particulates and gaseous pollutants, such as silica, Cr⁶⁺, Al₂O₃, SO₂, N₂O, CO, and hydrocarbons, into the atmosphere ². Workers

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involved in cement production and related activities are continuously exposed to these contaminants, which may exert adverse effects on various organ systems through passive inhalation, skin contact, or accidental ingestion^{1,2}. About 16% of annual global deaths from chronic obstructive pulmonary disease (COPD) are linked to occupational exposure to airborne particulates. In 2016, it was estimated that nearly 519,100 deaths worldwide were due to inhalation of hazardous dusts such as silica, coal dust, and asbestos^{3,4}.

Research has demonstrated links between cement dust exposure and systemic health changes⁵. For example, increased exposure is associated with inflammatory reactions and oxidative damage within critical organs, including the respiratory system, kidneys, and liver. Inhaled cement dust, rich in silica and chromium, can provoke immune and inflammatory responses, leading to conditions ranging from skin irritation and allergic reactions to bronchopulmonary illnesses and fibrotic changes in lung tissue⁶. Cement dust also contains heavy metals and trace elements that contribute to its toxic profile, raising concerns about cumulative and long-term effects in exposed populations⁷.

Cement dust exposure may compromise liver function. Although the acute effects may be minimal, studies reveal that chronic and repeated exposure can result in abnormalities in liver enzymes, with variations in Serum Glutamic Pyruvic Transaminase (SGPT), Serum Glutamic-Oxaloacetic Transaminase (SGOT), and alkaline phosphatase (ALP)⁸⁻¹⁰. Evidence from Bangladesh and Iraq points toward perturbations in these biomarkers, sometimes remaining within reference ranges but still indicating possible subclinical liver injury. Such findings highlight the adaptive and potentially reversible nature of some liver responses. Yet, they do not exclude the possibility of underlying chronic stress or damage, especially when exposure is prolonged or intense^{8,11}.

Animal and human investigations have shown that the composition of cement dust, including crystalline silica, calcium oxides, aluminum, and heavy metals such as chromium, can lead to oxidative stress, increased white blood cell counts, and persistent inflammation¹². These reactions may result in changes in the hematological profile and liver enzyme activities, impacting overall health. In particular, the role of silica and chromium in disrupting immune and liver cell function has been noted by previous studies, with some reporting

significant alterations in ALP, SGPT, and SGOT levels among workers, indicative of hepatic cell membrane impairment.^{11,13}

Workplace evaluation studies, particularly those conducted in Iraq and Bangladesh, have observed gaps in worker training, the use of personal protective equipment (PPE), and awareness of cement dust hazards^{8,11}. Many exposed workers lack adequate facemasks and undergo limited health screening, increasing the risk of adverse outcomes. These observations highlight an urgent need for strict regulation, surveillance, and health education to minimize exposure and promote safe working practices.¹⁴⁻¹⁶

The past research has reported liver dysfunction, especially as exposure duration increases, while some studies note minimal and statistically insignificant effects on key liver biomarkers^{8,13}. This discrepancy may arise from differences in study design, exposure levels, or population characteristics. However, the presence of even subtle changes in hepatic markers points to the necessity of regular health monitoring and preventive interventions in occupational settings. Despite the well-established respiratory effects of cement dust^{17,18}, its hepatotoxic potential remains less thoroughly explored. Therefore, this research aims to observe the effects of exposure to cement dust on liver enzymes in cement plant workers in Bangladesh.

Objectives of the Study

To assess the liver enzymes (SGPT, SGOT, and ALP) in workers exposed to cement dust in the cement plant of Bangladesh

Problem Statement

The toxic components of cement dust are small enough to enter the bloodstream and affect various organs. However, the low levels of inflammation may not immediately bring about permanent damage to the affected organs. Over time, the effects become visible. By the time they become clinically detectable, permanent damage has occurred, often leaving the workers to early retirement and a hefty health expenditure baggage. Such treatment is frequently neither affordable nor accessible for these individuals of the low-middle-income countries living in poverty¹⁵. As reported in previous literature, liver function changes (often subtle) have been noted with cement dust exposure^{8,11}. This research was conducted to determine whether liver function is altered upon exposure to this toxic dust

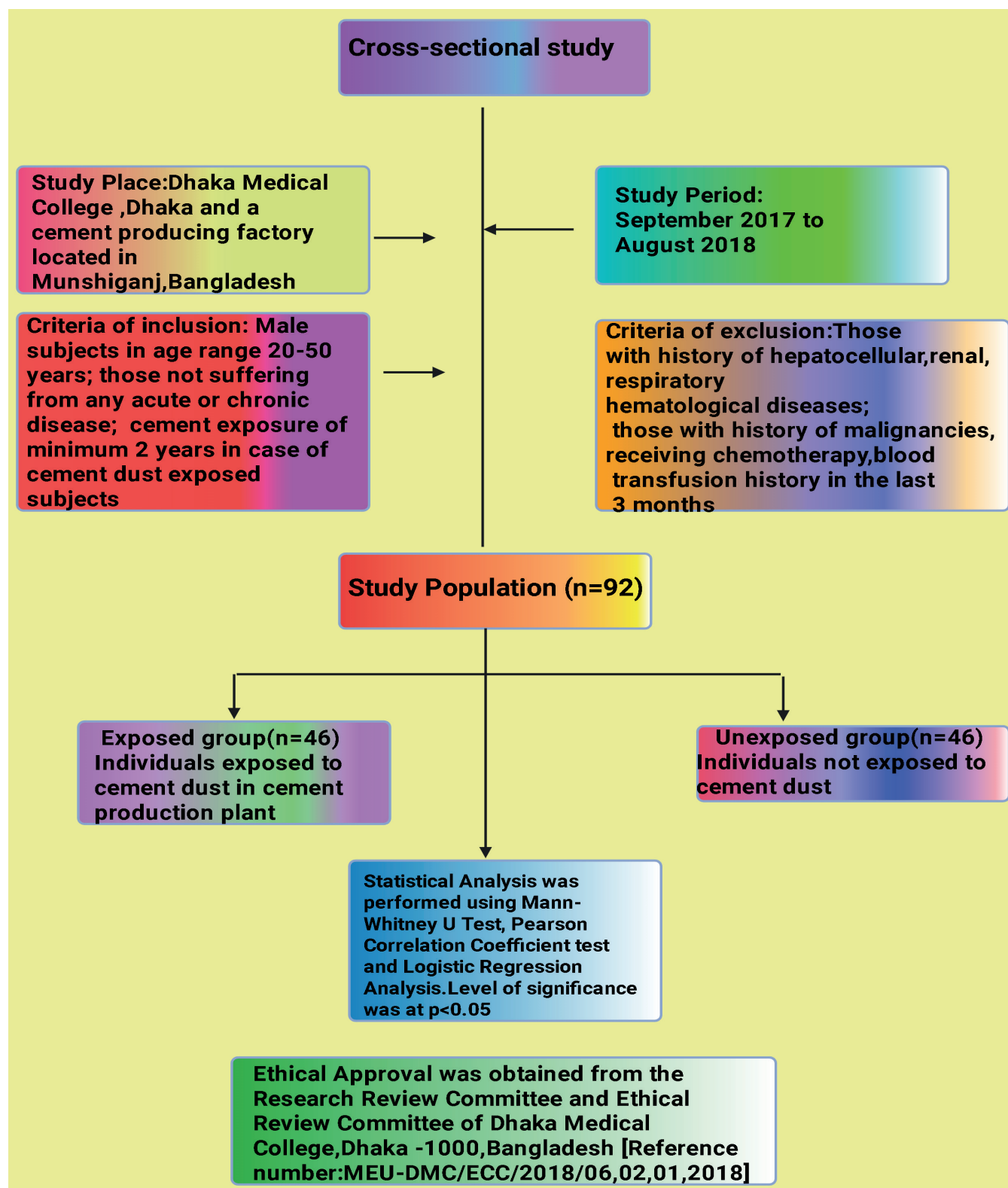


Figure 1: Materials and Methods of the study. This figure was drawn using the premium version of BioRender (<https://biorender.com/>, accessed on November 13, 2025), with license number WY28ZQPGOX.

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during work at the cement plant. If such changes are detected, this test may be included as part of a regular, affordable, and accessible health-monitoring routine for workers. Early detection of liver dysfunction can aid in the prevention of permanent health damage in the workers of a cement-producing plant.

MATERIALS AND METHODS

This cross-sectional study was conducted from September 2017 to August 2018 at Dhaka Medical College, Dhaka, Bangladesh, to evaluate the effects of occupational exposure to cement dust on liver function in male adults. The target population comprised 92 male participants who appeared to be healthy between the ages of 20 and 50 years. Of these, 46 individuals were employed at a Munshiganj-based cement production mill in the country, having been consistently exposed to cement dust for at least 2 years. The remaining 46 participants had no occupational exposure to cement dust and were residents of various localities across Dhaka city, providing a suitable control group. Sample size was calculated using a statistical formula from a previous manuscript¹⁹. The following flowchart illustrates the materials and methods [Figure 1].

Sampling Technique

Non-randomized purposive sampling was done for the research

Ethical Consideration

Participants were recruited using convenience sampling after approval from the relevant institutional ethical committee [Ethical Review Committee of Dhaka Medical College, Dhaka-1000, Bangladesh (Reference No.: MEU-DMC/ECC/2018/06, dated 02.01.2018)].

Participant recruitment as per the inclusion and exclusion criteria

All participants gave informed written consent prior to inclusion. Selection criteria for the exposed group mandated regular employment at the cement factory in roles involving direct contact with cement dust, such as crushing, milling, packing, and loading, for at least 8 hours per day, 6 days per week, which was in line with the previous study⁸. Controls were matched by age and gender, with exclusion of those with histories of chronic diseases, including liver disease, cardiovascular disease, hematological disorders, respiratory ailments, acute and chronic infection, and malignancies. Those with a blood transfusion history in the 3 months prior to

the onset of the study were excluded as well.

Questionnaire Development

The standard questionnaire of the Occupational Safety and Health Administration (OSHA)^{20,21} was used to develop the questionnaire of this research. The questionnaire was modified to consider the inclusion and exclusion criteria and the country's cultural background. A comprehensive history was taken from each participant, including demographic details, occupational background, residence, medical and family history, lifestyle habits (smoking and alcohol consumption history), and a history of drug intake such as anti-hypertensives, anti-allergic medications, anti-diabetic medications, steroids, Non-steroidal anti-inflammatory drugs, anticoagulants, and chemotherapy. Additionally, the travel history of the unexposed participants was recorded to ensure they had not been exposed to cement dust. General and systemic physical examination was also performed.

Three (3) ml of venous blood was collected aseptically from all participants. A blood draw was performed from the median cubital vein using sterile technique for laboratory assessment. The samples for liver function tests were placed in EDTA and plain tubes, stored at 4°C, and transported to the biochemistry laboratory within 4 hours to minimize sample degradation. All samples were processed uniformly to ensure reliability and comparability across groups. Hepatic function tests included determination of serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) utilizing automated biochemical analyzers following standardized protocols.

Sampling Technique

1. Sample Collection

- Blood samples (3 ml) were collected from the median cubital vein under strict aseptic conditions, using sterile disposable syringes.
- The skin was cleaned with spirit and gauze, and a tourniquet was applied 3 cm above the elbow to facilitate venipuncture.
- Drawn blood was split between plain and red-top test tubes for biochemical assays.

2. Sample Handling and Transport

- Blood tubes were immediately stored in a cold box at 4°C to preserve sample integrity.

- Samples were transported daily to the laboratory within 4 hours of collection, minimizing vibration and temperature fluctuations.
3. Serum Separation
 - Samples were centrifuged (at 3000 rpm for 5 minutes) to separate the serum from cellular elements.
 - Serum portions were aliquoted and stored at -20°C until biochemical evaluation.
 4. Biochemical Analysis of Liver Function
 - Liver function tests (LFTs) assessed included alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP).
 - Automated biochemistry analyzers and standardized enzyme assay kits were used to quantify these analytes under uniform conditions.
 - All measurements were performed in accordance with the manufacturer's protocols and quality control standards to ensure consistent results across samples.

Data Recording and Analysis

Data were entered into a pre-designed computer sheet and analyzed using SPSS version 26. Mann-Whitney U test, Pearson Correlation Coefficient test, and Logistic Regression Analysis were performed when applicable. Statistical significance was considered at $p < 0.05$. Data were presented as medians, interquartile ranges, box-and-whisker plots, Forest plots, and graphs. Careful attention was paid throughout the study to ethical conduct, voluntary participation, data confidentiality, and the right of each participant to withdraw at any time.

RESULTS

The study consisted of 92 male adult recruits, where the median age of the participants in the exposed group was 32 years, with a median cement dust exposure of 7 years. The median age of the unexposed group was 30 years [Table 1]. Our respondents had serum SGPT levels of 32 (25.7-52.5) U/L in the exposed group and 37.5 (25-48.2) U/L in the unexposed group. A significant difference ($p < 0.001$) in serum SGPT levels was observed between the two groups [Figure 2]. Our respondents had serum SGOT levels of 18.5 (15.7-24.2) U/L in the exposed group and 19 (16-24) U/L in the unexposed group. A significant difference ($p < 0.001$) in serum SGOT levels

was observed between the two groups [Figure 3]. Our respondents had serum ALP levels of 65 (54-78) U/L in the exposed group and 60.5 (53.5-76.7) U/L in the unexposed group. A significant difference ($p < 0.001$) in serum ALP levels was observed between the two groups [Figure 4]. The Pearson Correlation Coefficient test showed a negative relation with serum SGPT ($r = -0.872$, $p < 0.001$) and SGOT ($r = -0.856$, $p < 0.001$) levels, and a positive relation with serum ALP level ($r = +0.962$, $p < 0.001$) in study subjects [Figure 5]. We observed that duration of exposure to cement dust > 7 years were independent risk factors for reducing serum SGPT (OR: 7.636; 95% CI: 1.952 to 29.87; $p < 0.001$) and SGOT (OR: 8.333; 95% CI: 2.132 to 32.575; $p < 0.001$) levels and elevating serum ALP (OR: 17.857; 95% CI: 2.568 to 64.179, $p < 0.001$) level of cement dust worker [Figure 6].

Table 1: Age and duration of exposure of the study subjects (N=92)

Variable	Exposed group	Unexposed group	p value
Age (Years)	32 (27-38.5)	30 (26.5-38)	0.534
Duration (Years)	7(5-10)	-	-

Notes: Data were expressed as median and Interquartile Range (IQR). Mann-Whitney Test: U test was performed. Notes: Mann-Whitney Test U test was performed. Exposed: 32 (25.7-52.5), Unexposed: 37.5 (25-48.2), $p < 0.001$.

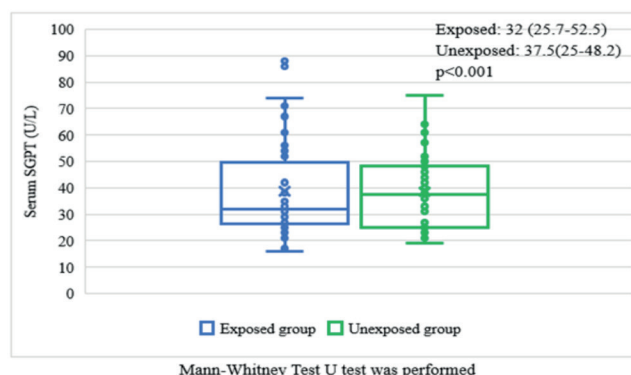


Figure 2: Box and whisker plot showing serum SGPT level of study subjects (N=92).

Notes: Mann-Whitney Test, U test was performed. Exposed: 18.5 (15.7-24.2), Unexposed: 19 (16-24), $p < 0.001$.

Illustration Credit: Mahmuda Abira.

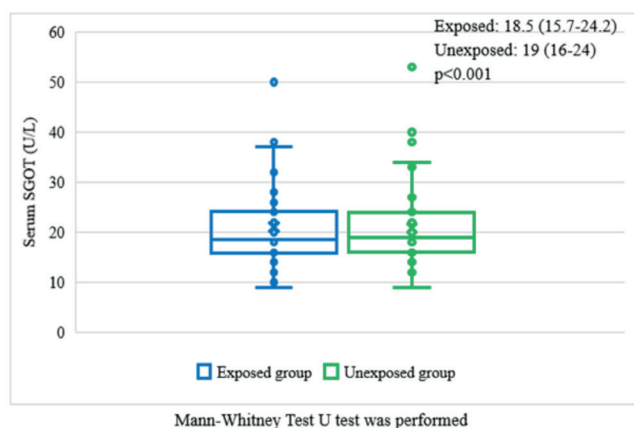


Figure 3: Box and whisker plot showing serum SGOT level of study subjects (N=92).

Notes: Mann-Whitney Test, U test was performed. Exposed: 65 (54-78), Unexposed: 60.5(53.5-76.7). $p < 0.001$.

Illustration Credit: Mahmuda Abira.

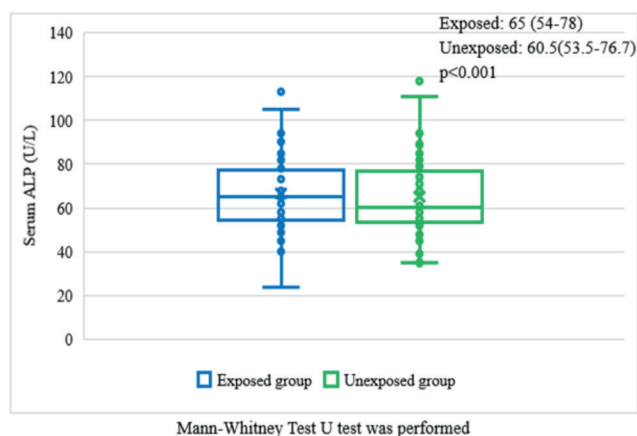


Figure 4: Box and whisker plot showing serum ALP level of study subjects (N=92).

Notes: SGPT: $r = -0.872$, SGOT: $r = -0.856$, ALP: $r = +0.962$, $p < 0.001$.

Illustration Credit: Mahmuda Abira.

DISCUSSION

The outcome of this research shows that cement dust-exposed recruits had significantly elevated Alkaline Phosphatase levels, while showing a significant reduction in Serum Glutamic Pyruvic Transaminase (SGPT) and Serum Glutamic-Oxaloacetic Transaminase (SGOT) levels. Even though the exact pathophysiology underlying the substantial rise in ALP in exposed subjects may not be fully elucidated, these alterations are indicative of subclinical hepatic stress following occupational exposure to cement dust¹³. The absence

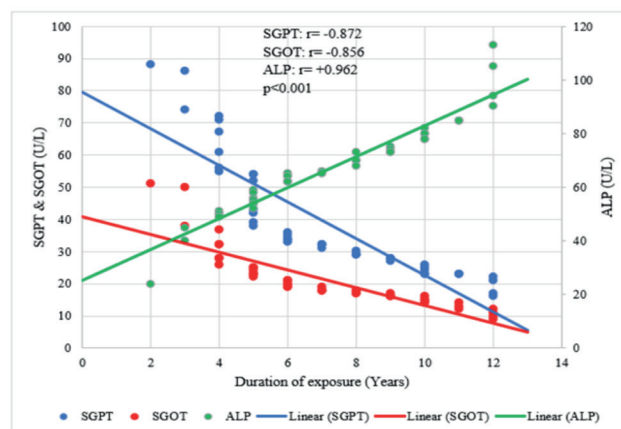


Figure 5: Pearson's Correlation Coefficient test showing a negative relation with serum SGPT and SGOT levels and a positive relation with serum ALP level of study subjects (n=46).

Notes: Odd Ratio (OR). $p < 0.001$.

Illustration Credit: Mahmuda Abira.

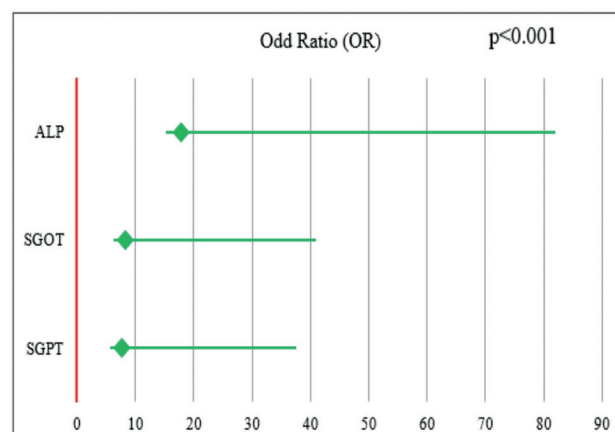


Figure 6: Forest plot displaying a significant association between duration of exposure and liver enzyme dysfunction (n=46).

Illustration Credit: Mahmuda Abira.

of a significant decrease in SGPT and SGOT may be due to the liver's adaptive mechanism, or the threshold for acute toxicity to cause an observable change in liver enzymes had not been crossed in the cement dust-exposed individuals. However, the significant alteration in ALP should be considered as a warning of possible hepatobiliary dysfunction and an indication for adopting intervention through dissemination of awareness and enforcement of the use of Personal Protective Equipment (PPE)^{8,23}.

Previous studies have reported a range of outcomes for liver function following occupational exposure to cement dust. In contrast, some have found results similar to our research, like those performed by Mojiminiyi et al., 2008 and Alameri et al., 2025 who reported no significant difference, or even lower enzyme levels, suggesting heterogeneity in risk related to factors such as exposure intensity, duration, use of protective equipment, genetic susceptibility, nutrition, and underlying health condition^{19,23}. Research performed by Oloruntoba et al., 2021 observed a reduction in SGPT, which may be a reflection of physiological adaptation to the toxic environment or could have resulted from the exposure levels not crossing the toxicity levels in the body²⁴. However, more extensive research is needed to explore the possible pathophysiology further and confirm these understandings. Another study by Akhter et al., 2022 reported significantly higher SGOT levels than in controls. Our research findings were in the same line; the alteration in the liver enzyme remained within normal range in their study, but in comparison to the unexposed subjects, the difference was significant. They suggested the change may be attributed to subclinical liver stress⁸.

With longer duration of exposure, the liver enzymes SGPT and SGOT showed a negative association, while a positive relationship was noted for ALP with duration of exposure. The disruption in liver enzymes over time may be due to oxidative stress, which may eventually lead to poor liver outcomes. Oxidative stress owing to inflammation caused by toxic components present within cement dust can lead to a deteriorating effect on the liver. An investigation done by Jasim et al., 2012 observed that the harmful elements of cement caused the formation of reactive oxygen species, which damaged liver cells and disrupted the normal hepatic physiology²⁵. Even though the liver enzymes were within normal limits in this study, the significant rise in ALP is suggestive of hepatobiliary changes possibly due to oxidative stress and subtle tissue remodeling, which, if unchecked, often leads to fibrosis²⁶.

Crystalline silica, a component of the respirable cement dust particles, may play a vital role in the process of oxidative stress, inflammation, and tissue remodeling. Respirable particles have an aerodynamic diameter of <3-4 μm and are therefore able to pass through into the blood^{26,27}. Crystalline silica increases the likelihood of disseminating to various organs, including the liver,

spleen, and bone marrow, via the lympho-hematogenous pathway. In organs like the liver, the silica particles may be taken up by macrophages, followed by the release of lysosomal enzymes that may bring about degradation alterations in the extracellular matrix (ECM), and fibrogenesis may occur^{26,28,29}. Other research works have suggested some mechanisms that may inflict liver damage due to exposure to silica. These include immunological disruption, oxidative or inflammatory pathway, epithelial-mesenchymal transition, and ECM remodeling³⁰⁻³².

In an experimental research performed on two-month-old CBA male mice, exposure to silica was reported to be linked to early and extensive occurrence of fibrotic process in the parenchyma of the liver and reduction of intracellular and cellular regeneration³³. A critical pathological event in the lung parenchyma of silicotic rats is the upregulation of Matrix Metalloproteinases (MMPs), including Gelatinase A (MMP-2) and B (MMP-9). These promote disruption of the basement membrane, which evolves into fibrosis³⁴. A similar rise in MMP-2 and MMP-9 was noted by Perez et al. 1999 and Scabilloni et al., 2005 in their experimental studies^{35,36}.

A study by Zawilli et al., 2014 on liver function in crystalline silica-exposed workers found overexpression of MMP-9 in exposed subjects, significantly higher than in non-exposed recruits. They also noted a positive correlation between MMP-9 levels and employment duration in workers exposed to silica ($r = 0.356$, $p < 0.001$)¹¹. Previous studies have found a correlation between MMPs and toxic liver damage, liver cirrhosis, and inflammation in chronic liver hepatitis^{37,38}. Matrix MMP-9 causes the degradation of type IV basement membrane collagen and promotes the early stages of tissue remodeling, a characteristic of liver diseases³⁹. The rise in MMP-9 in silica-exposed workers in the research by Zawilli et al., 2014, and its positive correlation with duration of exposure, suggest that this component of cement dust harms the liver²⁶.

Crystalline silica also acts as a pathogen-associated molecular pattern (PAMP), which affects macrophages (a component of innate immunity), leading to the death. Upon the death of macrophages, there is the release of cytokines like Interleukin 1 and Tumor Necrosis Factor, leading to liver tissue damage and fibrosis by stimulation of fibroblasts³⁰ [Figure 7].

Despite the absence of overt clinical disease, the significant between-group differences observed here

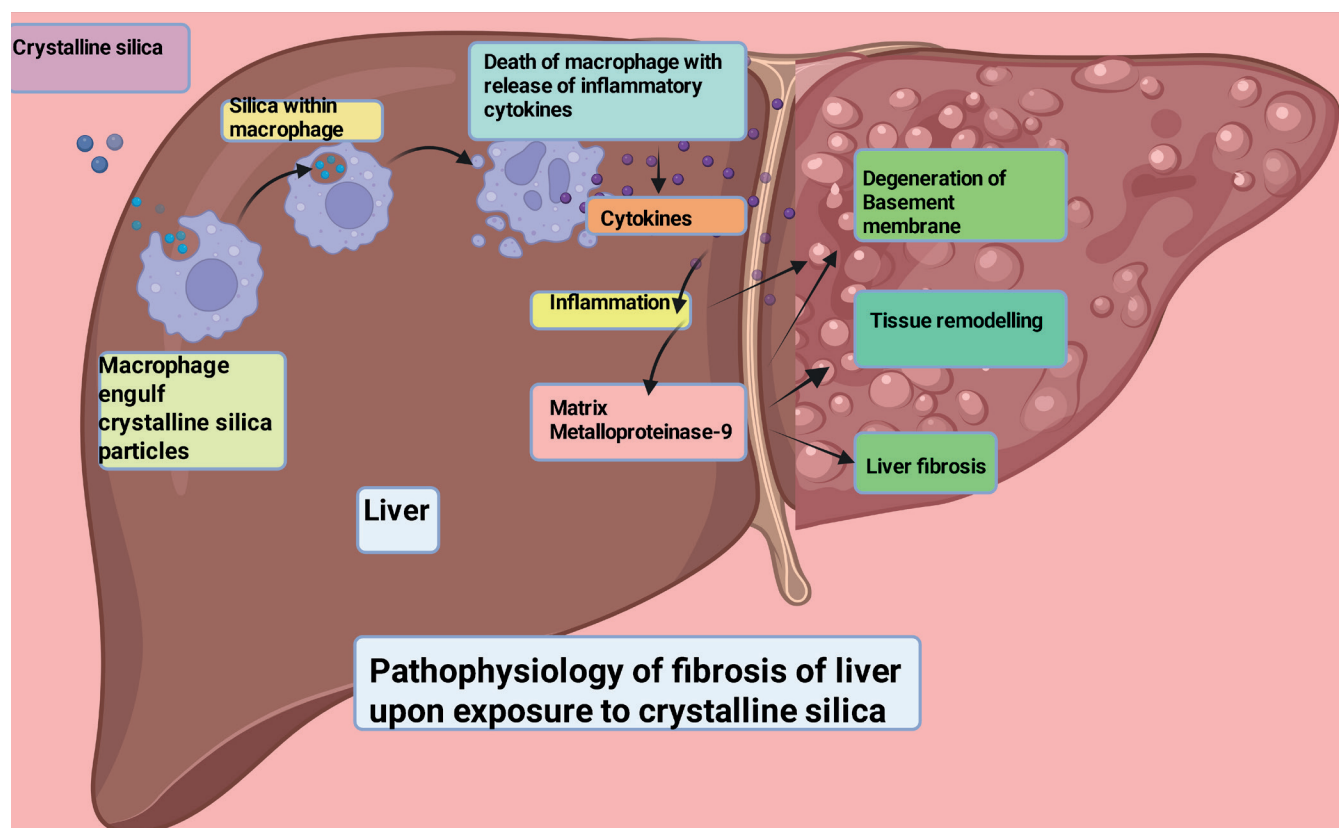


Figure 7: Impact of crystalline silica on the liver. Macrophages take up silica, leading to macrophage death and the release of cytokines that promote inflammation, MMP-9 formation, basement membrane degeneration, tissue remodeling, and liver fibrosis. This figure was drawn using the premium version of BioRender (<https://biorender.com/>), accessed on November 9, 2025, with license number UZ28Z43L83.

Illustration Credit: Rahnuma Ahmad.

in ALP stress the importance of regular monitoring. Persistent low-level enzyme elevations may act as early indicators for more severe hepatic dysfunction if exposure continues unchecked^{40,41}. Therefore, it is necessary for factories and occupational health authorities to implement regular liver function screening for workers, offer educational programs, enforce protective measures, and improve workplace ventilation^{8,42}.

Chronic occupational exposure to cement dust, even at levels not resulting in clinical disease, may be associated with subclinical elevations in liver function markers^{43,44}. This scenario warrants attention, as it signals possible early hepatic stress and highlights opportunities for preventive occupational health strategies before progression to irreversible tissue injury. Assessment of the type, degree, and source of

exposure is also necessary. Workplace safety needs to be emphasized and ensured, with sufficient ventilation and enforcement of rules requiring the use of personal protective equipment such as masks, gloves, protective goggles, and protective outfits^{45,46}. Continued research with larger sample sizes and extended follow-up will be crucial for establishing clear exposure thresholds, long-term risks, and effective safeguard measures for at-risk worker populations^{11,47}.

The outcomes of this study add to the growing body of evidence suggesting alterations in liver biomarkers in exposed individuals with both short- and long-term exposure to this toxic dust. The positive association of ALP with increasing duration of exposure points towards hepatobiliary impairment over time with chronic exposure to toxic components of cement dust^{8,13,25}. With growing exposure duration, the chemical

Key findings of the study

Occupational cement dust exposure causes significant health risks, especially affecting organs like the liver

Chronic inhalation of cement dust triggers inflammation and oxidative damage impacting multiple organ systems

Occupational exposure to cement dust may result in disruption of liver enzyme levels, indicating early hepatic stress in workers.

Cement dust components like silica and heavy metals induce oxidative stress and immune responses in the liver

Subclinical liver injury may be signaled by mild but significant elevations in ALT, AST, and ALP.

Cement dust causes systemic inflammation, affecting lungs, kidneys, and liver through inhalation and contact.

Regular health monitoring and safety measures are essential to prevent long-term liver damage

Improving workplace safety and protective equipment can significantly mitigate occupational health risks.

Figure 8: Key findings of the study. This figure was drawn using the premium version of BioRender (<https://biorender.com/>), accessed on November 9, 2025, with license number WZ28Z69Q8C.

Illustration Credit: Rahnuma Ahmad.

components of cement dust increase the possibility of hepatocellular damage, gradually leading to hepatic dysfunction and even permanent damage, such as fibrosis^{23,26}. As longer exposure duration may place workers at higher risk of hepatic dysfunction, regular health monitoring and the use of PPE are necessary²³.

Limitations of This Original Paper

This investigation was performed as a cross-sectional study in a single cement-producing plant, and therefore, the cause-and-effect association could not be established. The oxidative stress markers, MMP levels, could not be assessed due to time and financial constraints. The

effects of individual components of cement dust on liver function could be carried out. The concentrations of cement dust the workers were exposed to could not be measured due to financial and time limitations.

CONCLUSION

Although the liver enzymes remained within the normal range, there was a significant rise in ALP levels suggestive of subclinical hepatobiliary stress. A fall in SGPT and SGOT levels may be attributed to the body's adaptation to the toxic environment. The changes observed should be considered as a wake-up call for health monitoring more closely, spreading awareness among the workers and owners of the cement plants. The policymakers, both at the government level and by the owners of the cement plant, need to develop policies to ensure the use of PPE as well as allow rotational duty to make sure workers are not always working in the presence of a high-dust environment. Careful monitoring of Liver function may help prevent permanent health damage to the workers, allowing them to be in good health and work for a longer duration.

Future Research Recommendation

Large-scale, multicenter studies are needed to further establish the effects of cement dust on liver function. This study was conducted on workers with a maximum exposure of 12 years. Data needs to be collected and analyzed over a longer period of exposure to the dust. Regular health monitoring should include liver function tests to detect early signs of liver dysfunction. The workplace environment should be improved, including proper ventilation, measures to control dust levels, and

enforcement of the use of PPE while handling cement. Awareness needs to be raised among workers and factory owners about occupational health hazards. Finally, air quality regulations and safety standards must be developed to ensure a safe workplace environment—the principal findings of this paper, illustrated in Figure 8.

Consent for Publication

The author has reviewed and approved the final version and agrees to be accountable for all aspects of the work, including any accuracy or integrity issues.

Disclosure

Mainul Haque works in the editorial board of Bangladesh Journal of Medical Science. Rest of the authors declare that they do not have any financial involvement or affiliations with any organization, association, or entity directly or indirectly related to the subject matter or materials presented in this review paper.

Data Availability

Information for this review paper is taken from freely available sources.

Authorship Contribution

All authors contributed significantly to the work, whether in the conception, design, utilization, collection, analysis, or interpretation of data, or all these areas. They also participated in the paper's drafting, revision, or critical review, gave their final approval for the version that would be published, decided on the journal to which the article would be submitted, and made the responsible decision to be held accountable for all aspects of the work.

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