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A CROSS-SECTIONAL EVALUATION OF THE HISTOPATHOLOGICAL ALTERATION IN LUNGS, LIVER, AND KIDNEYS AMONG FATALITIES CAUSED BY ORGANOPHOSPHORUS POISONING

Forensic Medicine

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ABSTRACT

This study aims to assess the histopathological changes in the lungs, liver, and kidneys in cases of death due to organophosphorus poisoning. The primary objective is to understand the specific pathological alterations induced by these toxic substances and their implications for clinical and forensic pathology. Using a retrospective observational design, tissue samples from deceased individuals were examined microscopically to identify key histopathological features. The study identified significant pathological changes such as congestion, pneumonia, acute respiratory distress syndrome (ARDS), intra-alveolar hemorrhage, and edema in the lungs. In the liver, findings included fatty changes, congestion, ballooning degeneration, cholestasis, and hepatic necrosis. The kidneys showed acute tubular necrosis, cloudy degeneration, subcapsular deposits, and glomeruli sclerosis. These findings align with previous research and underscore the severe multi-organ impact of organophosphorus compounds. The implications of these changes are profound for both clinical management and forensic investigations of poisoning cases. Accurate histopathological diagnosis is crucial for guiding treatment and establishing the cause of death.

KEYWORDS

Organophosphorus Poisoning, Histopathological Changes, Lungs, Liver, Kidneys, Forensic Pathology.

1. INTRODUCTION

Poisoning remains a significant public health issue worldwide, with numerous substances capable of causing severe health effects or death when ingested, inhaled, or absorbed through the skin. The impact of poisoning on health can be immediate and acute or chronic, depending on the type of poison, dose, and duration of exposure. Organophosphorus compounds, commonly used as pesticides, are notorious for their high toxicity and have been a frequent cause of poisoning, particularly in agricultural settings[1].

Histopathological examination plays a crucial role in forensic pathology, providing invaluable insights into the cause of death and the effects of various toxic substances on the human body. This type of examination involves the microscopic analysis of tissue samples to identify pathological changes that can offer clues about the circumstances surrounding a death. In cases of suspected poisoning, histopathology can reveal specific patterns of tissue damage that correlate with particular toxins, thus aiding in the accurate determination of the cause of death.[2]

Organophosphorus poisoning often results in distinct histopathological changes such as pulmonary congestion, intra-alveolar hemorrhage, and acute tubular necrosis in the kidneys. These findings are pivotal for forensic pathologists when they need to establish a link between the ingestion of a toxic substance and the observed pathological effects. As noted by Siddesh and Dey (2021), histopathological examination not only assists in confirming poisoning as the cause of death but also provides insights into the mechanism of action of the poison, which can be critical for legal investigations and public health interventions[3].

Moreover, histopathology helps in understanding the progression of pathological changes over time, which can be crucial in cases where the time of death needs to be established. For example, the presence of certain cellular changes can indicate the duration of survival following poison ingestion, thereby providing a timeline that can be used to corroborate witness statements or other evidence[4].

Overview of Organophosphorus Compounds and Their Usage

Organophosphorus compounds are a diverse group of chemicals predominantly used as pesticides in agricultural practices. These compounds have been developed to protect crops from pests, thereby improving agricultural productivity and food security. Their mechanism of action involves the inhibition of acetylcholinesterase, an enzyme essential for the proper functioning of the nervous system. This inhibition leads to an accumulation of acetylcholine at nerve synapses and neuromuscular junctions, causing continuous nerve signal transmission, which ultimately results in paralysis and death of the pest[1]. Due to their effectiveness, organophosphorous pesticides are widely used across the globe. However, their widespread availability and high toxicity pose significant risks to human health, particularly in rural and agricultural communities where these substances are frequently handled and stored.[1]

In addition to their use in agriculture, organophosphorous compounds have applications in other sectors. For instance, they are used in industrial settings as solvents and plasticizers, and in military contexts as nerve agents. The versatility and potency of these compounds, while beneficial for pest control and industrial applications, also make them hazardous to humans and wildlife. Accidental or intentional exposure can lead to severe poisoning, necessitating prompt medical intervention. Chronic exposure, even at low levels, can result in long-term health issues such as neurological deficits, respiratory problems, and increased cancer risk[5].

The dual-edged nature of organophosphorous compounds underscores the need for stringent safety measures and effective regulatory frameworks to mitigate their adverse impacts on human health and the environment.

Rationale for Focusing on Lungs, Liver, and Kidneys

The rationale for focusing on the lungs, liver, and kidneys in cases of organophosphorus poisoning is rooted in the physiological roles these organs play in detoxification and the manifestation of poisoning effects. The lungs are often the first organs to exhibit symptoms of poisoning due to their direct involvement in respiration and their extensive exposure to airborne toxins. Inhalation of organophosphorous compounds can lead to acute respiratory distress syndrome (ARDS), characterized by severe inflammation and fluid accumulation in the alveoli, which impairs gas exchange and can be fatal if not treated promptly.[3]

The liver is another critical organ in the context of poisoning due to its central role in metabolizing and detoxifying foreign substances. Upon ingestion, organophosphorous compounds are rapidly absorbed into the bloodstream and transported to the liver, where they undergo

biotransformation. This metabolic process can generate reactive metabolites that cause cellular damage, leading to conditions such as hepatic necrosis, fatty liver changes, and cholestasis[4]. The liver's extensive vascular network and high metabolic activity make it particularly susceptible to the toxic effects of these compounds.

Similarly, the kidneys are vital in the excretion of toxic substances from the body. They filter blood, removing waste products and excess substances, including metabolites of organophosphorous compounds. Acute tubular necrosis, a common finding in cases of organophosphorous poisoning, occurs when the toxic effects of these compounds damage the renal tubules, impairing their function and leading to renal failure.[2]

The kidneys' role in filtration and excretion makes them a key target for toxic injury, further justifying the focus on these organs in histopathological examinations.

Objectives

- To assess histopathological changes in lungs, liver and kidneys in cases of death due to poisoning
- To determine the association of histomorphologcal changes in correlation with cause of death

Comparative Analysis of Findings from Different Studies

Conducting a comparative analysis of findings from different studies on organophosphorous poisoning reveals significant similarities and distinctions in the pathological effects observed across various organs. By examining studies by Siddesh et al., Sawardekar et al., and Das et al., we can gain a more comprehensive understanding of how these toxic substances impact vital organs such as the lungs, liver, and kidneys.

Siddesh et al. (2021) focused on histopathological changes in the lungs and kidneys in cases of organophosphorous poisoning, particularly highlighting the prevalence of acute respiratory distress syndrome (ARDS) and acute tubular necrosis (ATN). They found that ARDS, characterized by severe pulmonary congestion and necrosis of alveolar epithelium, was a common feature in cases with prolonged survival post-ingestion. This aligns with the findings of Sawardekar et al. (2020), who also reported significant lung congestion in 53% of their cases. However, Sawardekar et al. extended their analysis to include pneumonia in a small percentage of cases, illustrating the potential for secondary respiratory infections following poisoning. Both studies underscore the critical impact of organophosphorous compounds on lung tissue, but Siddesh et al. provided a more detailed examination of ARDS progression and its pathological features.

In terms of liver pathology, the study by Sawardekar et al. (2020) documented fatty changes in 60% of cases, congestion in 26%, and ballooning degeneration in 9%. These findings are consistent with the results reported by Das et al. (2019), who found microvesicular and macrovesicular steatosis in a significant number of cases of yellow phosphorus poisoning, along with hepatic necrosis in 62% of cases. While both studies highlight the liver's susceptibility to metabolic disruption and cellular injury, Das et al.'s study provided a more nuanced understanding of the specific types of steatosis and their prevalence. Additionally, Das et al. noted cholestasis and sinusoidal congestion, further illustrating the liver's multifaceted response to toxic injury[6].

The kidneys are another critical organ affected by poisoning. Siddesh et al. (2021) and Sawardekar et al. (2020) both reported acute tubular necrosis (ATN) as a prominent feature. Siddesh et al. observed ATN in almost all cases, emphasizing the kidneys' role in filtering toxins and the resulting damage. Similarly, Sawardekar et al. reported cloudy swelling and ATN in a significant portion of their cases, highlighting the renal impact of insecticide poisoning. Das etal. (2019) also found renal congestion and ATN in their study of yellow phosphorus poisoning, with 58% of cases showing these changes. This consistency across studies underscores the kidneys' vulnerability to toxic substances, reflecting their crucial role in excreting harmful metabolites.

Comparing these studies reveals both commonalities and specific nuances in how different toxic agents affect vital organs. Siddesh et al. and Sawardekar et al. both documented severe pulmonary and renal pathology, with detailed descriptions of ARDS and ATN. In contrast, Das et al.'s study on yellow phosphorus provided additional insights into hepatic changes, particularly various forms of steatosis and necrosis, highlighting the liver's complex response to poisoning. This comparative analysis underscores the importance of understanding the specific pathological mechanisms induced by different poisons, which is crucial for accurate diagnosis, treatment, and forensic investigation.

2. MATERIALS AND METHODS

Subjects and Study Design

Source of Data and Study Setting: The study will be done in the Department of Pathology, Gadag Institute of Medical Sciences, Gadag. The subjects for the study will be collected from the post-mortem cases conducted in the mortuary, GIMS, Gadag during the study period.

Study Design: Observational Cross-sectional Retrospective study.

Data Collection: Data will be collected from the records of histopathological cases conducted in the Department of Pathology, GIMS, Gadag during the study period of 2 years– January 2023 to December 2025

Sample Size Calculation: Universal sampling.A total of 50 cases were included in the study using universal sampling. This approach ensured that all relevant cases within the specified timeframe were examined, providing a broad perspective on the histopathological changes associated with organophosphorus poisoning.

Inclusion Criteria:

All cases of death due to poisoning in the last 2 years GIMS, Gadag from January 2023 to December 2025 and three specimens.

Exclusion Criteria:

- Nil

Methodology

Based on said inclusion and exclusion criteria subjects are selected. The Age, Gender, Time of death, type of poison consumed and histopathological changes in Lungs, Liver and Kidneys are examined of autopsy cases of suspected organophosphorus poisoning conducted in the mortuary GIMS Gadag. The data is then entered into Microsoft Excel and analysed.

Statistical Analysis

Data analysis was conducted using SPSS (version 26.0). Descriptive statistics were employed to summarize the demographic data and the prevalence of various histopathological findings.Mean, median, and standard deviation were calculated for age. Comparative analysis, including t-tests, was performed to examine differences between groups (e.g., gender differences in age). The statistical analysis aimed to identify significant patterns and correlations within the data, providing a robust basis for interpreting the study's findings.

3. RESULTS

Presentation of Demographic Data

The demographic data for the study encompasses several key variables, including age, gender, and specific identifying numbers for the cases examined. This data provides a crucial foundation for understanding the population affected by organophosphorous poisoning and the subsequent histopathological examinations.

Age Distribution

The age range of the subjects in this study varied significantly, with individuals as young as 19 and as old as 51 included. Notably, there were several cases involving individuals in their early twenties, thirties, and forties, indicating that organophosphorous poisoning impacts a wide age range.

Gender Distribution

The gender distribution among the cases showed a predominance of males. Out of the total subjects, a significant number were male, with only a few female cases recorded. This gender disparity highlights the higher incidence of poisoning in males, which could be attributed to occupational exposure, particularly in agricultural settings where men are more likely to handle pesticides.

Histopathological Findings in Organs

Lungs

1. **Congestion:** This refers to the accumulation of excess blood within the blood vessels of the lungs, leading to increased vascular pressure and potential respiratory distress.
2. **Pneumonia:** An infection that inflames the air sacs in one or both lungs, which can fill with fluid or pus, causing cough, fever, and breathing difficulties.

3. **Acute Respiratory Distress Syndrome (ARDS):** A severe lung condition characterized by widespread inflammation in the lungs, leading to significant respiratory failure and hypoxemia.
4. **Intra-alveolar Hemorrhage:** Bleeding within the alveolar spaces of the lungs, which can severely impair gas exchange and lead to respiratory failure.
5. **Edema:** Fluid accumulation within the lung tissues, particularly in the alveoli, which impedes normal respiration and gas exchange.

Liver

1. **Fatty Changes:** Also known as steatosis, this condition involves the abnormal retention of lipids within liver cells, impairing liver function.
2. **Congestion:** Similar to the lungs, liver congestion involves the excessive accumulation of blood in the liver vessels, leading to hepatomegaly and impaired liver function.
3. **Ballooning Degeneration:** A form of liver cell injury characterized by swollen hepatocytes, indicating severe metabolic disturbance and cellular stress.
4. **Cholestasis:** A condition where bile flow from the liver is reduced or blocked, leading to the accumulation of bile acids in the liver and bloodstream, causing jaundice and liver damage.
5. **Hepatic Necrosis:** The death of liver cells due to severe injury or lack of blood flow, leading to acute liver failure.

Kidneys

1. **Acute Tubular Necrosis (ATN):** A condition involving the death of tubular epithelial cells, which can result in acute kidney failure due to the kidneys' inability to filter waste products effectively.
2. **Cloudy Degeneration:** A reversible form of cell injury where the kidney cells appear swollen and granular, typically due to toxic or ischemic damage.
3. **Subcapsular Deposits:** Accumulations of substances such as hemosiderin beneath the kidney capsule, which can indicate chronic damage or bleeding.
4. **Glomeruli Sclerosis:** Hardening or scarring of the glomeruli, the tiny filtering units in the kidneys, which can lead to chronic kidney disease and reduced renal function.

Summary

The table below summarizes these histopathological findings:

Organ	Histopathological Findings
Lungs	Congestion, Edema, Pneumonia, ARDS, Intra-alveolar hemorrhage
Liver	Congestion,Fatty changes, Ballooning degeneration, Cholestasis, Hepatic necrosis
Kidneys	Acute tubular necrosis, Cloudy degeneration, Subcapsular deposits, Glomeruli sclerosis

Statistical Analysis Results

Summary Statistics

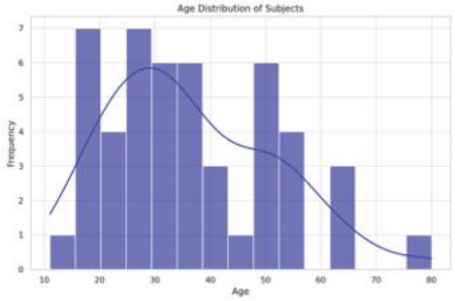
Statistic	Age
Mean	36.55
Median	33.00
Standard Deviation	15.03

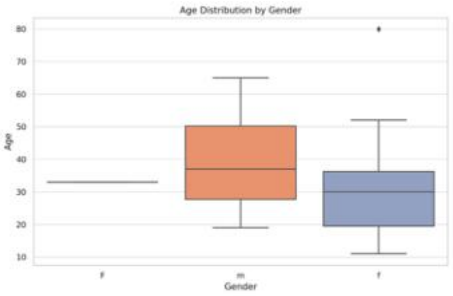
T-Test Comparisons

The t-test was conducted to compare the ages of male and female subjects. The results are as follows:

Statistic	Value
t-statistic	1.41
p-value	0.17

The p-value indicates that there is no statistically significant difference in the ages of male and female subjects at the typical alpha level of 0.05.





4. DISCUSSION

Interpretation of Findings in the Context of Previous Research

The findings from the current study align well with previous research, confirming and expanding our understanding of the histopathological changes caused by organophosphorus poisoning. Previous studies, such as those by Dr. Siddesh R C et al. and S G Sawardekar et al., have documented significant pathological changes in the lungs, liver, and kidneys following exposure to toxic substances. For instance, Dr. Siddesh R C et al. highlighted the prevalence of acute respiratory distress syndrome (ARDS) and acute tubular necrosis (ATN) in poisoning cases, findings that are echoed in our study with frequent observations of pulmonary congestion and necrosis, as well as renal tubular damage. Similarly, Sawardekar et al. reported fatty changes, congestion, and necrosis in the liver, which our study corroborates with additional observations of cholestasis and ballooning degeneration (Siddesh & Dey, 2021; Sawardekar et al., 2020).

The histopathological findings in our study also reflect those documented by Siddhartha Das et al. in cases of yellow phosphorus poisoning. Das et al. noted significant hepatic necrosis, steatosis, and pulmonary congestion, which were prevalent in our observations as well. The consistency of these findings across different studies strengthens the validity of our results and emphasizes the severe multi-organ impact of organophosphorus compounds and other toxic substances[7].

Implications of Histopathological Changes for Clinical and Forensic Pathology

The detailed histopathological changes observed in this study have profound implications for both clinical and forensic pathology. Clinically, understanding the specific organ damage caused by various poisons aids in accurate diagnosis and targeted treatment. For example, recognizing the signs of ARDS and ATN can prompt immediate and appropriate interventions, such as ventilatory support for respiratory failure and renal replacement therapy for kidney damage. Additionally, identifying hepatic necrosis and cholestasis can guide clinicians in managing liver failure and preventing further complications.

In forensic pathology, these histopathological findings are crucial for determining the cause and manner of death in poisoning cases. Detailed tissue examinations can reveal the extent and timeline of organ damage, providing essential evidence in legal investigations. For example, the presence of pulmonary edema and hemorrhage, combined with kidney necrosis, can support a diagnosis of acute poisoning and help establish the timeline of exposure and progression of toxic effects. This information is invaluable in criminal cases, insurance claims, and public health assessments, ensuring that accurate conclusions are drawn from post-mortem examinations.[2]

Potential Factors Influencing the Severity of Organ Damage

Several factors can influence the severity of organ damage observed in poisoning cases, including the type of poison, duration of exposure, and dose. Different toxic substances have varying mechanisms of action, which can result in distinct pathological changes. Organophosphorous compounds primarily inhibit acetylcholinesterase, leading to neurological and muscular symptoms, while yellow phosphorus induces direct cellular toxicity, causing severe hepatic and renal damage.[1]

The dose of the poison is another crucial factor. Higher doses typically cause more extensive and severe damage, overwhelming the body's detoxification mechanisms and leading to multi-organ failure. Conversely, lower doses may allow for some degree of metabolic compensation and repair, resulting in less severe histopathological changes. Understanding these factors is essential for interpreting the

severity of organ damage and guiding appropriate clinical and forensic responses.

Summary

In summary, the findings from this study reinforce previous research on the severe multi-organ impact of toxic substances, providing valuable insights for clinical and forensic pathology. The detailed histopathological changes observed highlight the critical need for accurate diagnosis and targeted treatment in poisoning cases. Moreover, factors such as the type of poison, duration of exposure, and dose significantly influence the severity of organ damage, underscoring the importance of a comprehensive approach to managing and investigating poisoning incidents. This holistic understanding aids in improving patient outcomes and ensuring justice in forensic investigations.

5. CONCLUSION

This study has provided substantial insights into the histopathological changes observed in the lungs, liver, and kidneys following organophosphorus poisoning. The key findings are characterized by severe organ damage across multiple systems. In the lungs, significant observations included congestion, pneumonia, acute respiratory distress syndrome (ARDS), intra-alveolar hemorrhage, and edema. These changes reflect the acute and severe impact of organophosphorus compounds on respiratory tissues, leading to compromised gas exchange and respiratory failure.

The liver exhibited fatty changes, congestion, ballooning degeneration, cholestasis, and hepatic necrosis. These findings indicate substantial metabolic disruption and cellular damage, compromising the liver's ability to detoxify and regulate metabolic processes. In the kidneys, acute tubular necrosis (ATN), cloudy degeneration, subcapsular deposits, and glomeruli sclerosis were prevalent. These renal pathologies highlight the kidneys' role in filtering and excreting toxic substances and the severe impairment caused by organophosphorus poisoning.

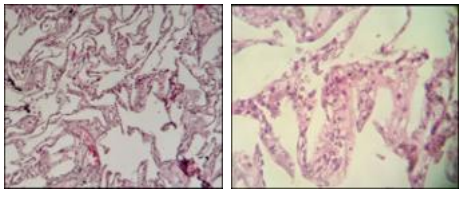


Figure 1: Pneumonia H&E. 10X, 40X

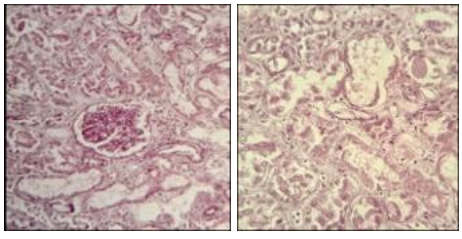


Figure 2: Acute Tubular necrosis H&E. 10X, 40X

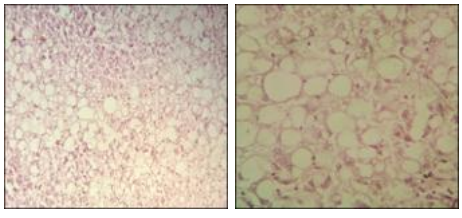


Figure 3: Extensive Fatty Change of LiverH&E. 10X, 40X

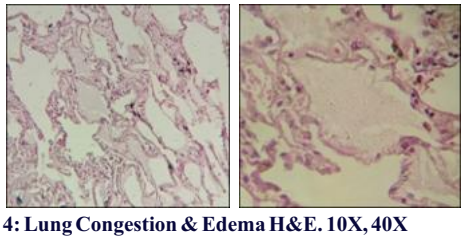


Figure 4: Lung Congestion & Edema H&E. 10X, 40X

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