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Original Research Article

Clinicopathological Alterations in Oral Tissues Following Pesticide Poisoning: An Autopsy-Based Evaluation

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Pesticide poisoning, Postmortem changes, Oral manifestations, Histopathology, Degenerative changes.

Abstract

Introduction: Pesticide poisoning remains a major public health issue, especially in developing countries like India, where it accounts for 95% of fatal poisoning cases. According to the WHO, pesticides are the most common means of suicide, with millions of incidents reported annually. Both suicidal and homicidal incidents are worsened by easy access and low cost. The oral cavity is crucial to forensic investigations since oral intake is the most frequent exposure route. **Materials & methods:** This cross-sectional study evaluated clinical and histopathological changes in oral tissues caused by pesticide ingestion. Autopsied tissues of gingiva (n=22) and tongue (n=19) from pesticide-ingested deaths were compared with 10 control samples. **Results:** Gingiva showed statistically significant changes in epithelium (p value= 0.027) and connective tissue (p value= 0.003) with prominent degenerative changes. Clinically, aluminium phosphide and paraquat showed severe tissue alterations in the oral cavity. Notably, this is the first study to examine the postmortem histopathological changes of pesticide poisoning in gingiva. **Conclusion:** As the gingiva shows significant changes and it is easily accessible, it could be the tissue assessed for identifying toxic pesticide poisons. Oral autopsy is a vital forensic technique in medico-legal investigations for determining the cause of death in cases of insufficient body retrieval.

1. Introduction

Forensic Poisoning has emerged as a major non-communicable disease epidemic in the 21st century.[1] Pesticide poisoning, in particular, is a significant cause of morbidity and mortality, especially in developing countries.[2,3] In India, where nearly 75% of the population is engaged in agriculture, pesticide poisoning accounts for approximately 95% of poisoning-related fatalities.[3,4] According to World Health Organization estimates in 2020, pesticides remain the most common method of suicide worldwide, with an estimated 385 million cases of unintentional acute pesticide poisoning occurring annually, resulting in nearly 11,000 deaths.[5] Overall, poisoning constitutes 63% of all toxic exposures, affecting 65% of adults and 22% of pediatric cases.[6]

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Poisoning has become a prevalent non-communicable disease epidemic in the 21st century.[1] Pesticide Action Network (PAN) India, in a report released in March 2024, stated that 339 pesticide actives are registered under India's Insecticides Act, 1968, of which approximately 100 are herbicides (about 30%), and 118 are classified as Highly Hazardous Pesticides (HHPs).[7] n India, the most prevalent pesticides associated with deadly outcomes were organophosphates, aluminium phosphide, and paraquat. Oral ingestion is the most common route of pesticide exposure, followed by inhalation and skin contact.[8] Pesticide poisoning was previously accidental, but its widespread availability, low cost, and unrestricted sale have led to a rise in suicidal and homicidal incidents. Adolescents who attempt self-harm through ingestion tend to suffer more severe poisoning than young children, who are often exposed accidentally through ingestion, skin contact, or inhalation.[9,10]

The oral cavity is often regarded as a window to the body's internal changes, which reflects nearly all systemic variations resulting from disease, injury, or death.[11] Gingival tissue is one of the most accessible and structurally stable oral tissues, and its integrity during the early post-mortem period makes it particularly valuable for forensic assessment.[12] However, no previous study has explored its histological changes in pesticide-related deaths. Hence, the present study was undertaken to evaluate microscopic alterations in the gingiva, along with the tongue, in pesticide poisoning cases to aid in the forensic interpretation of the cause of death.

2. Materials and Methods

This cross-sectional study was conducted in the Department of Oral Maxillofacial Pathology and Oral Microbiology at KLE Vishwanath Katti Institute of Dental Sciences, in collaboration with the Department of Forensic Medicine & Toxicology at KLES, Dr. Prabhakar Kore Hospital & Medical Research Centre, after obtaining the approval from the institutional ethical committee. All fatal cases of pesticide poisoning, whether from deliberate or accidental ingestion, involving any age group or gender, that were brought to the Prabhakar Kore Hospital & Medical Research Centre between January and October 2024 were included in this study.

Study population: Regardless of age or gender, all deceased individuals with the history of poisoning consumption within a 15-hour period were included. Deaths from other causes and decomposing bodies were excluded. Deceased individuals, regardless of age or gender, who did not have a history of pesticide exposure provided the control samples.

Study group: Postmortem tissue samples were obtained from the deceased individuals with a history of pesticide ingestion (Gingiva; n=22, Tongue; n=19).

Control group: Postmortem tissue samples were collected from deceased individuals without a history of pesticide ingestion (n=10).

Consent: Those who met the qualifying requirements were only included in the study after their families gave their informed consent.

Data Retrieved from the Patient Charts and inquest forms: History regarding the type of pesticides and demographic details were retrieved from the patient charts. A total of 41 tissue samples - 22 from the gingiva and 19 from the tongue were analysed. The age of the individuals in the study group ranged from 15 months to 57 years, with a mean age of 30.75 ± 15.62 years, whereas the control group comprised individuals aged 18 to 55 years, with a mean age of 35.2 ± 10.6 years with a slight male predominance (56%). Organophosphorus compounds were the most common poison detected (63%), followed by paraquat (22%), zinc phosphide (5%), aluminium phosphide (5%), and Agroneem (5%).

Sample Collection: Photographs were taken both intra and extra orally prior to oral autopsy. Following this, tissue samples were collected from the gingiva & tongue. Samples were immediately preserved in formalin for 24 hours. Routine histopathological processing was done followed by Haematoxylin and Eosin (H & E) staining. No special stains or immunohistochemical (IHC) techniques were applied, as the study focused on general histopathological alterations. Three independent oral pathologists analysed the sections for the histopathological alterations in the study samples and control samples. To minimize observer bias, interobserver variability was addressed through the consensus method, wherein all three observers reviewed the slides collectively and arrived at a unanimous agreement on the final diagnosis and grading of histological features.

Clinical analysis: Extra and intraoral examinations were done to appreciate any changes that are induced by various pesticide poisons.

Histopathological analysis: The changes in the epithelium and connective tissue were assessed. Sequential analysis of the following histological

changes of the biopsies of all individuals were carried out as follows-

1. Epithelial changes
2. Shredding, Pseudoepitheliomatous Hyperplasia, Atrophy, Melanin, Intercellular Bridges.
   a. Cytoplasmic changes: Intracellular vacuolization, intracellular oedema, and intracellular oedema.
   b. Nuclear features: clumping of chromatin, presence/absence of distinct nuclear outline, karyolysis, pyknosis, karyorrhexis, vacuolation, and homogenization.
   c. Separation of epithelium and connective tissue.
3. Connective tissue changes
   a. Fibroblasts vacuolation, Shredding of fibres, Eosinophilia, Homogenisation, Presence of inflammation, Shrinkage.
4. Any other findings
   a. Degenerative changes of muscles, salivary gland, nerve tissues, blood vessels, adipose tissues.

The degree of histological changes in the post-mortem samples was correlated to various pesticide poisons. Data was tabulated in an Excel spreadsheet, followed by evaluation of Frequencies and percentages.

3. Results

On clinical examination, the oral cavity revealed various intraoral & extraoral manifestations due to consumption of pesticide poisons. Individuals who consumed paraquat had a necrotic ulceration on their lip with black eschar, bleeding, and external discoloration **(Fig. 1A).** Individuals with aluminium phosphide consumption exhibited a necrotic, discoloured, and ulcerated area along with the presence of sloughing, fibrosis, and tissue breakdown **(Fig. 1C).** Areas exhibiting mucosal blanching, especially on the labial mucosa, with a pale, fibrotic look, represent ischemic damage changes in people who consume zinc phosphide and organophosphorus compounds **(Fig. 1B & D).** Histopathological examination, analyzed for the postmortem changes induced by various pesticide poisons, showed prominent histopathological changes in epithelium, connective tissues, and their components in both gingiva and tongue samples compared to the controls.

Histopathological postmortem changes observed in gingiva due to pesticide ingestion: On examination, gingiva exhibited epithelial changes such as pseudoepitheliomatous hyperplasia, shredding **(Fig. 2A & B),** atrophy, prominent intercellular bridges, and melanin deposition in the basal layers of the epithelium. Intracellular vacuolization was observed in the cytoplasm. Likewise, nuclear changes such as clumping of chromatin, presence of distinct nuclear outline, karyolysis, pyknosis, karyorrhexis, vacuolation, homogenization, and melanin deposition are observed in the tissues of the gingiva **(Fig.2 C&D).**

Fig. 1A to D: Photomicrographs showing oral manifestations induced by various pesticide poisons. (A) Paraquat poisoning, (B) Organophosphorus poisoning, (C) Aluminium phosphide, (D) Zinc phosphide.

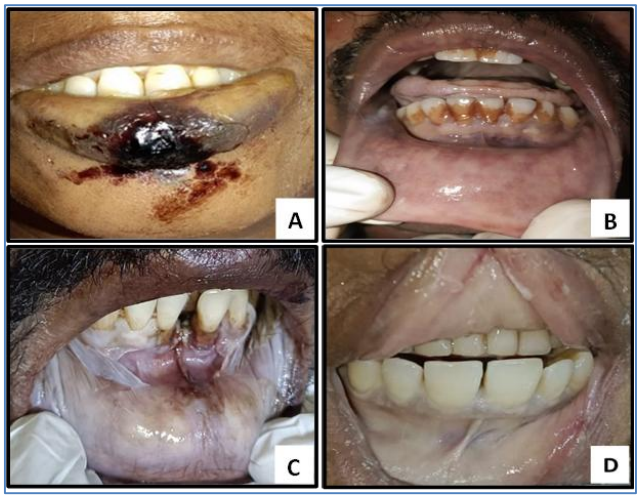
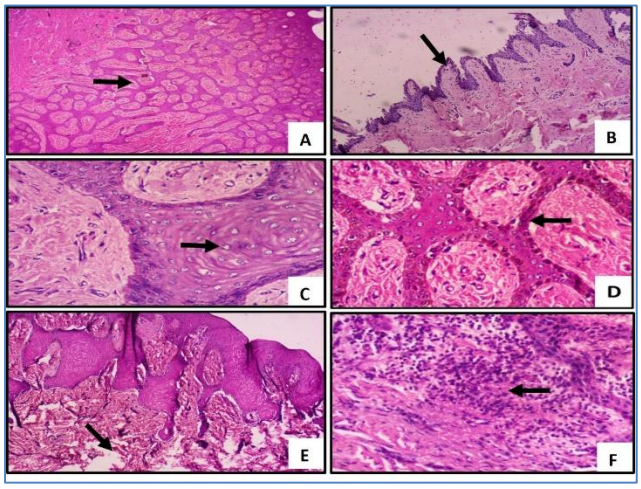


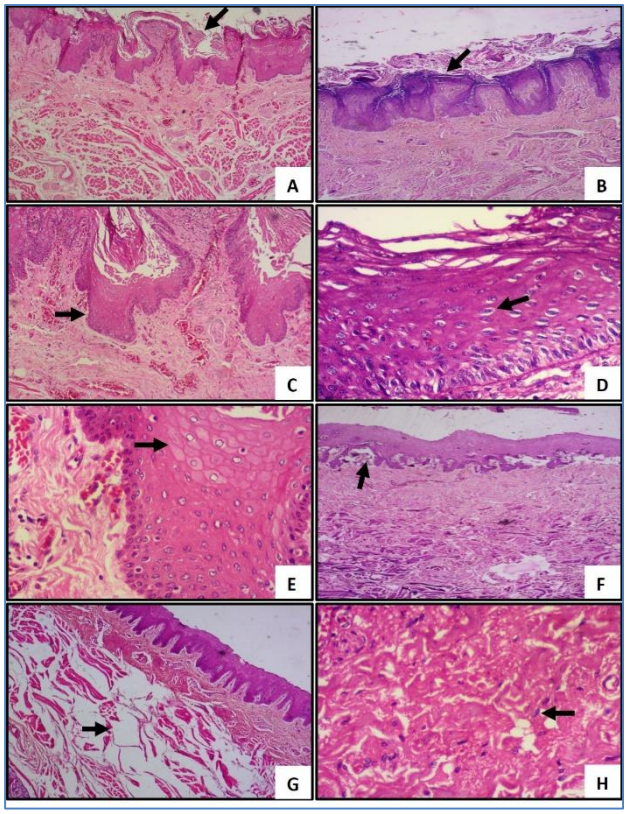
Fig. 2 A to F: Microscopic section of gingiva induced by pesticide poisons showing Pseudoepitheliomatous hyperplasia, Shredding (A&B). Cytoplasmic & Nuclear changes (C&D) Shredding of collagen fibres (E), Presence of inflammation (F).



Histopathological postmortem changes observed in the tongue due to pesticide ingestion: On examination, tongue exhibited almost similar histopathological changes compared to the gingiva. The epithelial changes include shredding, stripping **(Fig. 3A & B)**, pseudoepitheliomatous hyperplasia,

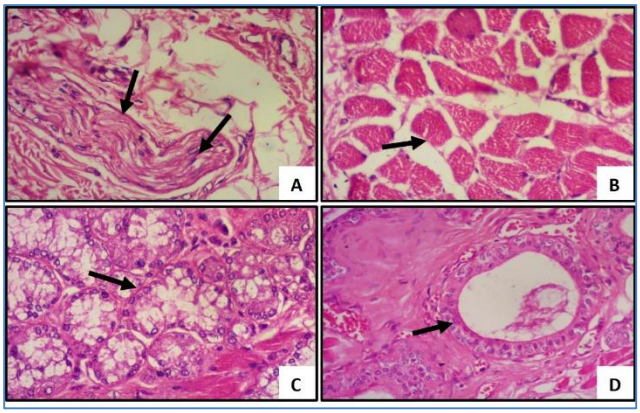
and prominent intercellular bridges **(Fig. 3C).** Cytoplasmic changes such as intracellular vacuolization, intracellular edema, & intercellular edema were observed in the cytoplasm. Likewise, nuclear changes such as clumping of chromatin, presence of distinct nuclear outline, karyolysis, pyknosis, karyorrhexis, vacuolation and homogenization were seen **(Fig.3D & E)**. Separation between epithelium & connective tissue **(Fig. 3F)** are also noted. In connective tissues, changes included shredding of collagen fibres **(Fig. 3G)**, vacuolization of fibroblasts **(Fig. 3H)**, embedding of the fibroblasts within the collagen fibers, eosinophilia, homogenization, and shrinkage along with chronic inflammation predominantly of lymphocytes.

Fig. 3 A to H: Microscopic section of tongue induced by pesticide poisons showing Stripping & Shredding of epithelium(A&B) pseudoepitheliomatous hyperplasia, prominent intercellular Bridges (C) Cytoplasmic changes & Nuclear changes (D & E) Separation between Epithelium & Connective tissue(F) Shredding of Connective tissue (G) vacuolisation of fibroblasts (H).



Degenerative changes observed in Connective tissue Components: Degenerative changes such as loss of architecture, vacuolization of the nucleus and cytoplasm of nerve fibers **(Fig. 4A)**, muscle fiber degeneration with loss of striations **(Fig. 4B)**, loss of architecture of the salivary gland acini along with vacuolization of acinar cells **(Fig. 4C).** Dilation of salivary ducts **(Fig. 4D)** and loss of architecture along with congestion & dilation of blood vessels were also noted. These changes were not found in control samples.

Fig. 4 A to D: Microscopic section of connective tissue components induced by pesticide poisons showing Degenerative changes such as loss of architecture, vacuolization of nucleus and cytoplasm of nerve fibers (Fig.4A), muscle fibres degeneration with loss of striations (Fig.4B), loss of architecture of the salivary gland acini along with vacuolisation of acinar cells (Fig.4C) Dilation of salivary ducts (Fig.4D).



However, on comparison with the control group, i.e., deceased individuals without a history of pesticide ingestion, gingiva samples showed statistically significant changes in both the epithelium (p= 0.027) and connective tissues (p = 0.003). Whereas, the tongue samples did not show many significant changes in epithelium and connective tissue compared to control samples but exhibited prominent degenerative changes in the connective tissue components **(Table 1).** An independent analysis was conducted to assess histopathological postmortem changes across various types of pesticide poisoning, aiming to determine the possibility of identifying specific pesticides. However, no statistically significant association was observed, possibly due to the unequal distribution of cases among the pesticide types **(Table 2).**

Statistical analysis: Statistical analysis was performed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics (frequency and percentage) were calculated, and group comparisons were done using the Chi-square test. A p-value < 0.05 was considered statistically significant. No correction for multiple comparisons was applied due to the limited number of comparisons. Three independent oral pathologists analysed the sections for histopathological alterations in both study and control samples. To minimize observer bias, interobserver variability was addressed through the

Table 1: Postmortem Histopathological Changes of Gingiva & Tongue in Pesticide Poisoning Cases. Note: F- Frequency, P – Percentage, X^2 - Chi-square, p – P value, *P<0.05 – Statistically significant.

Histological domain	Observed Changes	Gingiva				Tongue			
		Control Samples		Study Samples		Control Samples		Study Samples	
		F	P (%)	F	P (%)	F	P (%)	F	P (%)
1.Epithelium	Stripping	-	-	-	-	0	0	12	63.16
	Shredding	0	0	13	59.09	0	0	16	84.21
	Pseudoepitheliomatous hyperplasia	10	90	14	63.64	1	8	11	57.89
	Atrophy	0	0	8	36.36	0	0	11	57.89
	Melanin	6	55	18	81.82	-	-	-	-
	Intercellular Bridges	6	55	14	63.64	2	17	9	47.37
		X^2 - 10.90, p - 0.027*				X^2 - 3.315, p - 0.345			
2.Nucleus	Chromatin clumped	2	18	18	81.82	2	17	7	36.84
	Karyolysis	9	81	19	86.36	6	50	17	89.47
	Karyorrhexis	4	36	16	72.73	6	50	16	84.21
	Pyknosis	6	55	12	54.55	3	25	12	63.16
	Nuclear Outline	11	100	21	95.45	12	100	19	100.00
	Intracellular Vacuolization	8	72	17	77.27	7	58	11	57.89
	Intranuclear vacuolisation	-	-	-	-	7	58	15	78.95
	Homogenization	0	0	12	54.55	0	0	12	63.16
	Intercellular Edema	-	-	-	-	2	17	12	63.16
	Intracellular Edema	-	-	-	-	1	8	14	73.68
		X^2 - 9.98, p - 0.125				X^2 -9.024, p - 0.4349			
3.Separation of Epithelium &Connective Tissue		0	0	0	0	0	0	7	36.84
4.Connective Tissue	Fibroblast embedded within collagen fibres	2	18	19	86.36	-	-	-	-
	Vacuolization of fibroblast	6	55	12	54.55	5	42	17	89.47
	Shredding of Fibres	0	0	12	54.55	0	0	9	47.37
	Eosinophilia	0	0	4	18.18	0	0	7	36.84
	Homogenization	0	0	17	77.27	0	0	7	36.84
	Presence of inflammation	8	73	10	45.45	0	0	7	36.84
	Shrinkage of Connective tissue	0	0	14	63.64	0	0	9	47.37
		X^2 - 25.23, p - 0.003*				X^2 - 9.655, p - 0.085			
5. Any Other Findings	Muscles	-	-	-	-	4	33	14	73.68
	Salivary Gland	-	-	-	-	0	0	8	42.11
	Nerve Tissue	-	-	-	-	4	33	14	73.68
	Blood Vessels	-	-	-	-	4	33	18	94.74
	Adipose Tissue	-	-	-	-	1	8	9	47.37
		-				X^2 - 4.112, p - 0.390			

consensus method, wherein all three observers reviewed the slides collectively and arrived at a unanimous agreement on the final diagnosis and grading of histological features.

4. Discussion

This study underscores the importance of clinical and histopathological examination in evaluating postmortem changes due to pesticide ingestion. Our study is the first to examine the postmortem histopathological changes from autopsied tissue samples of gingiva with ingested pesticide poisoning. In cases of poisoning, post-mortem histopathological examinations are typically conducted on systemic organs such as the stomach, liver, and kidneys. These analyses help confirm the cause of death through qualitative and quantitative tests, as these organs are primarily involved in the absorption and elimination of the poison.[13]

However, in cases where only a partially recovered body is available, analysing systemic effects becomes challenging due to the loss of vital organs. In such situations, the oral cavity, being the first organ to come into direct contact with ingested poisons and one of the most protected areas, plays a crucial role. Histological examinations help assess tissue cell conditions and architecture, as exposure to chemicals or physical factors, including death, can lead to structural alterations in the tissues.[14]

Gingiva and tongue, being potential sources of tissue samples, were utilized for histopathological examination in individuals who had consumed pesticides in our study. These tissues remain stable against environmental effects because of their placement within the oral cavity, which is normally shielded from external influences. Control samples comprised oral tissues from deceased individuals who had not ingested with pesticides.

Table 2: Postmortem Histopathological Changes in Gingiva & Tongue Induced by Specific Pesticides.

Histological domain	Features	Gingiva					Tongue				
		OPC	PQ	ZP	AP	AG	OPC	PQ	ZP	AP	AG
1.Epithelium	Stripping	-	-	-	-	-	8	2	0	1	1
	Shredding	9	5	1	0	0	8	4	2	2	0
	Pseudo- epitheliomatous hyperplasia	7	3	2	1	1	6	2	2	0	1
	Atrophy	5	2	0	0	1	6	3	1	1	0
	Melanin	8	6	2	1	1	-	-	-	-	-
	Intercellular Bridges	8	3	1	1	1	5	1	1	1	1
		X^2 - 5.95, **p** - 0.99					**X^2** – 9.88, **p** - 1.00				
2. Nucleus	Chromatin Clumped	10	4	2	1	1	4	0	1	1	1
	Karyolysis	10	5	2	1	1	9	4	2	1	1
	Karyorrhexis	9	4	2	1	0	8	4	1	2	1
	Pyknosis	6	2	3	1	0	7	2	1	1	1
	Nuclear Outline	11	5	3	1	1	10	4	2	2	1
	Vacuolization	9	3	3	1	1	10	4	2	2	1
	Homogenization	6	4	1	1	0	6	2	1	2	1
	Intercellular Edema	-	-	-	-	-	6	2	1	2	1
	Intracellular Edema	-	-	-	-	-	6	4	2	1	1
		X^2 - 5.41, **p** - 1.00					**X^2** – 4.28, p - 1.00				
3. Separation of Epithelium & Connective Tissue		-	-	-	-	-	4	1	1	1	0
		-					**X^2** – NA, **p** - NA				
4. Connective Tissue	Fibroblast embedded within collagen fibres	9	6	3	0	1	-	-	-	-	-
	Vacuolization of fibroblasts	8	1	2	1	0	9	3	2	2	1
	Shredding of fibres	7	4	1	0	0	5	3	0	1	0
	Eosinophilia	2	1	0	1	0	3	1	2	1	0
	Homogenization	10	3	2	1	1	4	1	2	1	0
	Presence of inflammation	5	2	1	1	1	3	3	0	1	0
	Shrinkage	7	5	1	0	1	5	2	1	1	0
		X^2 - 14.3, **p** - 0.9					**X^2** - 9.70, **p** - 0.97				
5. Any Other Findings	Muscles	-	-	-	-	-	7	2	2	2	1
	Salivary Gland	-	-	-	-	-	5	1	1	1	0
	Nerve Tissue	-	-	-	-	-	10	1	2	2	0
	Blood vessels	-	-	-	-	-	10	3	2	2	1
	Adipose Tissue	-	-	-	-	-	6	2	0	1	0
		-					**X^2** – NA, **p** - NA				

Note: X^2 - Chi-square, p – P value, OPC – Organophosphorus Compound, PQ – Paraquat, ZP – Zinc Phosphide, AP – Aluminium Phosphide, AG – Agroneem $^*P<0.05$ – Statistically significant.

In the present study, organophosphorus poisoning emerged as the most prevalent type of pesticide used. Organophosphates block acetylcholinesterase, leading to excess acetylcholine and cholinergic toxidrome, affecting nicotinic, muscarinic, and CNS receptors. Symptoms appear rapidly through inhalation, ingestion, or skin absorption, and prolonged exposure can cause respiratory failure and death.[15-17] In contrast, Paraquat primarily targets the lungs and kidneys, accumulating in cells through energy-dependent transport. It generates reactive oxygen species, leading to alveolitis, pulmonary fibrosis, and renal tubular necrosis. It causes direct damage on contact with the mucosal lining of stomach or intestines and mouth.[18] Agroneem (Azadirachtin), derived from neem seeds, disrupts insect growth by inhibiting ecdysone. Though mainly an insecticide, human exposure can trigger vomiting, metabolic acidosis, leucocytosis, encephalopathy, and organ damage, with fatal cases showing fatty liver infiltration and mitochondrial dysfunction.[19] Similarly, Aluminium phosphide (ALP) poisoning has severe systemic effects. It releases phosphine gas upon ingestion, which inhibits oxidative phosphorylation, causing cellular hypoxia, circulatory collapse, and multi-organ failure. Unlike Agroneem, which disrupts metabolism gradually, Aluminium phosphide leads to rapid cardiovascular failure.[20] Zinc phosphide, a common rodenticide, also generates phosphine gas upon ingestion. Once absorbed into the bloodstream, it affects the liver and lungs, leading to metabolic disruption and respiratory failure, similar. However, its activation depends on stomach acid.[21]

Clinical examination in our study indicated that poisoning from Paraquat and Aluminium phosphide resulted in more severe toxic effects, causing notable mucosal changes in the oral cavity, including ulceration, erosion, and sloughing due to their direct contact toxicity, generation of reactive oxygen species or phosphine gas, and systemic oxidative or hypoxic injury to metabolically active tissues.[16,18] The

histopathological alterations observed—such as epithelial shredding, cytoplasmic vacuolization, nuclear pyknosis, and karyorrhexis—reflect a common underlying mechanism of oxidative stress–induced cellular injury. Pesticides such as organophosphorus compounds, paraquat, and aluminium phosphide generate reactive oxygen species (ROS), which cause mitochondrial dysfunction, lipid peroxidation, and protein denaturation, ultimately leading to cellular degeneration and necrosis. These effects disrupt epithelial integrity and connective tissue architecture, producing the characteristic degenerative and inflammatory changes noted in the present study. [22] The connective tissues also exhibited shredding of collagen fibers, vacuolization of fibroblasts, embedding of fibroblasts within collagen fibres, eosinophilia, homogenization, and shrinkage of connective tissues when compared to the control samples. Similarly, Satyanarayanan et al., who investigated the histopathological effects of chlorinated hydrocarbon pesticides on the tissues of common carp, reported shrinkage of connective tissue in the kidney.[23] Although conducted in fish, these findings are relevant as they support the universal mechanism of oxidative stress–induced structural damage, which is also evident in mammalian and human tissues. Separation between the epithelium and connective tissue was also observed, likely resulting from the degradation of basement membrane proteins, including laminin and collagen IV, caused by oxidative stress induced by organophosphate pesticides.[24]

Degenerative changes in the connective tissue components were observed in all pesticide poisoning cases. Congestion and dilation of blood vessels found are due the degeneration and damage to the endothelial cells.[25] The present study showed degeneration and loss of striations in muscle fibres. These results were comparable to those of Wecker L et al who investigated the histological effects of the organophosphate pesticide Parathion on human skeletal muscle fibres. The study found that the diaphragm and intercostal muscles in particular showed signs of nerve ending necrosis and degeneration. The muscle fibre necrosis is attributed to prolonged inhibition of cholinesterase (ChE) by organophosphates, leading to acetylcholine (AcH) accumulation and excessive muscle stimulation, causing cell damage.[26] The present study also showed degeneration and loss of architecture of the salivary gland acini along with vacuolization of acinar cells. All the specimens of ingested poisons revealed loss of architecture and vacuolization of nucleus and cytoplasm of nerve fibres. These results are comparable to those of Sowmiya et al.[8] However, no degenerative alterations were seen in the control samples. Even though the current study found significant changes in the epithelium and connective tissue, as well as degenerative changes in connective tissue components that support the identification of the cause of death, the distribution of the specific pesticide poisons is not equal, making it impractical to identify specific pesticide poisons through histopathological changes. Future studies with balanced sample distribution and molecular correlation could help identify diagnostic markers for specific poisons. There are various studies on toxicology and great future scope in the toxicological research.[27,28] With technological advancement and innovative approaches the Forensic toxicology is evolving and helpful to aid in administration of justice.[29,30]

5. Conclusion

The postmortem histopathological alterations of pesticide poisoning in gingiva are examined for the first time in our study. Both the gingiva and tongue have noticeable histopathological alterations. When compared to normal tissues, gingiva exhibited more pronounced alterations than tongue in cases of pesticide ingestion. Ingestion of pesticides also caused degenerative alterations in connective tissue components. Organophosphorus compound was found to be the most common pesticide compared to the other poisons. Clinically, aluminium phosphide and paraquat showed severe alterations in the oral cavity. Since the gingiva is readily accessible and exhibits notable alterations, it could be the tissue assessed for the detection of harmful pesticide poisons. In situations of inadequate body retrieval, oral autopsy serves as a crucial forensic tool for establishing the cause of death, highlighting its significance in medico-legal investigations. Future studies with larger, multi-centric sample sizes are recommended to strengthen statistical power and validate the diagnostic value of oral tissues in pesticide poisoning cases.

Ethical Clearance: IEC approval is taken from the Institutional Ethical committee.

Contributor ship of Author: All authors equally contributed. **Conflict of interest:** None to declare.

Source of funding: None to declare.

References:

1. Kandasamy G, Ramasamy K. Retrospective autopsy analysis on pattern of fatal cases of poisoning. MedPulse. Int J Forensic Med. 2019; 9(2):22–4.
2. Varghese P, Erickson TB. Pesticide poisoning among children in India: The need for an urgent solution. Glob Pediatr Health. 2022; 9: 2333794X221086577.
3. Aghera VJ, Pipaliya KN, Dabhi DM, Mangal HM, Varu PR, Manvar PJ. Study of gross mucosal findings of stomach in cases of fatal poisoning: An autopsy study. J Indian Acad Forensic Med. 2014; 36(1):26–30.
4. Awasthi PM, Verma S, Dev R, Bajpai A. Trend of fatal poisoning in Kanpur: A two-year autopsy-based study. Int J Res Med Sci. 2018; 6: 259–63.
5. Moebus S, Boedeker W. Case fatality as an indicator for the human toxicity of pesticides—a systematic scoping review on the availability and variability of severity indicators of pesticide poisoning. Int J Environ Res Public Health. 2021; 18(16):8307.
6. Mittal C, Singh S, Kumar-M P, Varthya SB. Toxicoepidemiology of poisoning exhibited in Indian population from 2010 to 2020: a systematic review and meta-analysis. BMJ open. 2021; 11(5):e045182.
7. Pesticide Action Network (PAN) India. Highly Hazardous Pesticides and Weedicides in India: Status and Concerns. PAN India; 2024. Available from: https://www.pan-india.org/
8. Rao RS, Vinesh E, Rajkumar C, Nichat P, Karthick BP, Chandra G, et al. Histopathological changes in oral tissues induced by pesticide poisoning: a pilot study. J Contemp Dent Pract. 2022; 22(11):1302-7.
9. Singh SP, Aggarwal AD, Oberoi SS, Aggarwal KK, Thind AS, Bhullar DS, et al. Study of poisoning trends in North India—a perspective in relation to world statistics. J Forensic Leg Med. 2013; 20(1):14–8.
10. Ahmad A, Chaudhry SH, Farooq U, Waheed I, Junaid A, Ali A. Pattern of self-poisoning and toxicity in suicidal deaths presenting for autopsy at a teaching hospital of the cosmopolitan city of Pakistan. J Ayub Med Coll Abbottabad. 2023; 21(1):55–60.
11. Arthanari A, Doggalli N, Arun M, Rani S. Oral manifestations of poisons in view of forensic odontology: A review. Medico Legal Update. 2020; 20(4):7–13.
12. Yadav AB, Angadi PV, Kale AD, Yadav SK. Histological assessment of cellular changes in postmortem gingival specimens for estimation of time since death. J Forensic Odontostomatol. 2015; 33(1):19–26.
13. Sutay SS, Tirpude BH. Pattern of histopathological changes of liver in poisoning. J Indian Acad Forensic Med. 2008; 30(2):63–8.
14. Adhikari SK, Chowdhuri S, Mukhopadhyay P. A study on the histopathological changes in cases of pesticide poisoning and proposed diagnostic algorithm. Indian J Forensic Med Toxicol. 2021; 15(2):96–102.
15. Robb EL, Regina AC, Baker MB. Organophosphate toxicity [Internet]. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2025 Jan [updated 2023 Nov 12; cited on 18 Apr 2025]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK558981/
16. Chaudhari VA, Das S, Devnath GP. Fatal suicidal poisoning: A record-based study in South India. J Forensic Med Sci Law. 2022; 11(2):547–52.
17. Manigandaraj G, Visnu RJ, James RJ. Profile of Deaths due to Poisoning, Autopsied in Chennai – A Cross Sectional Study. J Forensic Med Sci Law. 2025; 34(1):10-15. doi: 10.59988/jfmsl.vol.34issue1.3
18. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. Br J Clin Pharmacol. 2011; 72(5):745–57.
19. Iyyadurai R, Surekha V, Sathyendra S, Paul Wilson B, Gopinath KG. Azadirachtin poisoning: A case report. Clin Toxicol (Phila). 2010; 48(8):857–8.
20. Agrawal VK, Bansal A, Singh RK, Kumawat BL, Mahajan P. Aluminum phosphide poisoning: Possible role of supportive measures in the absence of specific antidote. Indian J Crit Care Med. 2015; 19(2):109–12.
21. Dolan E, Güzel A, Çiftçi T, Aycan E, Çelik F, Çetin B, et al. Zinc phosphide poisoning: A case report. Hum Exp Toxicol. 2008; 27(4):341–3.
22. Torres-Sánchez ED. Impacts of pesticides on oral cavity health and ecosystems: A review. Environ Sci Pollut Res Int. 2023; 30(3):5982–90.
23. Satyanarayanan S. Histopathological changes due to some chlorinated hydrocarbon pesticides in the tissues of *Cyprinus carpio*. IOSR J Pharm. 2012; 2(6):60–6.
24. Ghaneialvar H, Razi M, Malekinejad H, Moayeri A, Sadeghi N, Najafi G. The protective effect of alpha-lipoic acid on the expression of collagen IV, renal function, and oxidative stress induced by diazinon in the renal parenchyma of rats. Environ Sci Pollut Res Int. 2021; 28(6):6789–98.
25. El-Bendary HM, Shaker MH, Saleh AA, Negm SE, Khadey ME, Hosam Eldeen FA. Histopathological changes associated with exposure of male mice to profenofos and chlorpyrifos. Annu Res Rev Biol. 2014; 4(5):766–77.
26. Wecker L, Mrak RE, Dettbarn WD. Evidence of necrosis in human intercostal muscle following inhalation of an organophosphate insecticide. J Environ Pathol Toxicol Oncol. 1985; 6(2):171–5.
27. Deokar RB, Patil SS. Avenues in Forensic Medicine. J Forensic Med Sci Law 2023;32(2):1-3.
28. Deokar RB, Patil SS. Research Publication: Forensic Perspective. J Forensic Med Sci Law. 2024;33(2):1-4.
29. Keche AS, Bhargava DC, Vidua RK, Keche HA. Environmental Toxicology- Introduction & Legislation. J Forensic Med Sci Law. 2024;33(2):51-56. doi: 10.59988/jfmsl.vol.33issue2.9
30. Deokar RB, Dere RC, Patil SS. Looking at Recent Evolution in Toxicology Through Legal Lens. J Indian Acad Forensic Med. 2025;47(1):7-11.