J Indian Acad Forensic Med. 2021 Apr-Jun; 43(2): 135-139
doi:10.5958/0974-0848.2021.00035.X

ORIGINAL ARTICLE

Histopathological changes in lungs, liver and kidneys in cases of death due to agricultural poisoning in a tertiary care centre: A cross sectional study

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Abstract
Medico legal deaths due to agricultural poisoning claim a substantial number of lives in Andaman and Nicobar Islands. Respiratory failure is the most common complication of OP poisoning leading to death. Histopathological studies of viscera in organophosphate poisoning cases help the autopsy surgeon in ascertaining out the secondary causes of death. Microscopy of three visceral organs-lungs, liver and kidneys of 40 cases of organophosphate poisoning were studied and the changes were correlated with the duration of survival post consumption. By knowing the exact histopathological changes in these organs, it is easier to attribute death to a failure of one of these organs as a consequence of organophosphate consumption. Knowing the rapidity of involvement of an organ is beneficial to the treating physician as well thereby helping him reduce the morbidity and hence mortality.

Keywords
Organophosphate poison; Histopathology; Lungs; Liver; Kidneys; Cause of death

Introduction

Organophosphates constitute a heterogeneous category of chemicals specifically designed for the control of pests, weeds or plant diseases. Their application is still the most effective and accepted means for the protection of plants from pests, and has contributed significantly to enhance agricultural productivity and crop yields.[1] Their common availability renders organophosphate insecticide poisoning a worldwide health issue with a high fatality rate, especially the suburban and rural population. Most of the pesticide poisoning and subsequent deaths occur in developing countries following a deliberate self-ingestion of the poison.[2] It is roughly estimated in India that 5 to 6 people per lakh population die due to poisoning every year and the most common cause of poisoning in India and other developing countries is organophosphorus compounds.[3] Medico legal deaths including OP poisoning claim a substantial number of lives in Andaman and Nicobar Islands. Respiratory failure is the most common complication of OP poisoning leading to death. In every centre carrying out medico legal work, autopsies of poisoning cases form a sizeable group and the autopsy surgeon is required to give his opinion regarding the final cause of death which in most cases can only be determined after looking into the chemical analysis report from the Forensic Science Laboratory and the histopathological reports of the viscera sent to the Department of Pathology. In most of the cases death may occur immediately due to respiratory centre and muscle paralysis after consuming poison or may be delayed for days or weeks together. Delayed causes of death may be due to ARDS, respiratory paresis, liver failure and renal failure. In the latter cases the determination of exact cause of death may be difficult as external appearance may not give any clue and the internal examination may sometimes not reveal anything much on gross examination. Histopathological examination can reveal such pathologies in major organs like lungs, liver and kidneys where the poison acts, gets absorbed and is finally eliminated. This study was conducted with an aim to study the progressing effects of the insecticide on organs and to give a much clearer opinion regarding the cause of death.

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Article History
Received: 6th August, 2020; Accepted: 3rd May, 2021

Materials and Methods

This study was carried out in the Department of Forensic Medicine and Toxicology in collaboration with the Department of Pathology, ANIIMS, Port Blair, Andaman and Nicobar Islands for a period of 1 year with a sample size of 40 cases. It was ensured that in all 40 cases of ingestion of agricultural poisons, the three organs, lungs, liver and kidneys were collected and fixed in 10% formalin and sent for histopathological examination to Department of Pathology. The routine viscera were sent to the FSL for chemical analysis. Duration of survival in hours was noted in all cases. Cases which were treated in the hospital with a doubtful history of agricultural poison consumption were excluded from the study.

Results

The duration of survival post consumption has been divided into 3 categories: those who survived for less than 24 hours (<1 day), 24 hours to 72 hours of survival (1-3 days) and more than

72 hours of survival post consumption (>3 days). Out of the 40 cases, 6 cases (15%) survived for a duration of less than 24 hours,8 cases (20%) survived for a duration between 24 and 72 hours and 26 cases(65%) survived for a maximum duration; i.e more than 72 hours as shown in Table 1.

Table 1: Duration of survival (N=40)

Duration of Survival	n
<24 hours	6
24-72 hours	8
>72 hours	26

Table 2: Histopathological changes in the liver (N=40)

HPE findings	n	%
Congestion	21	52.5
Fatty changes	8	20
Centrilobular Necrosis	4	10
Alcoholic Hepatitis	5	12.5
Sinusoidal dilation	2	5

HPE = histopathological examination

Table 3: Comparing the changes in the liver with the duration of survival

	Duration of Survival		
HPE findings	<24 hours	24 -72 hours	>72 hours
Congestion	6	4 (50%)	11 (42.3%)
Fatty changes	0	2(25%)	6 (23%)
Centrilobular Necrosis	0	0	4 (15.3%)
Alcoholic hepatitis	0	2(25%)	3 (11.5%)
Sinusoidal dilation	0	0	2 (7.69%)
Total	6	8	26

HPE = histopathological examination

Table 4: Histopathological changes in the lungs (n=40)

HPE findings	n	%
Congestion	40	100
Suggestive of ARDS	38	95
Changes S/o Bronchopneumonia	2	5

HPE = histopathological examination

Table 5: Comparing the changes in the lungs with the duration of survival

	Duration of Survival		
HPE findings	<24 hours	24-72 hours	>72 hours
Congestion	0	0	26
Suggestive of ARDS	0	0	38
Changes S/o Bronchopneumonia	0	0	2

HPE = histopathological examination

Table 6: Histopathological changes in the kidneys (N=40)

HPE findings	N	%
Congestion	40	100
Suggestive of ATN	34	85

HPE = histopathological examination; ATN = acute tubular necrosis

Table 7: Comparing the changes in the kidneys with the duration of survival

	Duration of Survival		
HPE findings	<24 hours	24-72 hours	>72 hours
Congestion	40	+	+
Suggestive of ATN	0	8	26

HPE = histopathological examination; ATN = acute tubular necrosis; + = indication that congestion was associated with features suggestive of ATN

Table 8: Comparison between two studies on the HPE findings in the liver HPE findings

HPE findings	Present Study	Sutay and Tirpude[8]
Congestion	52.5%	46.51%
Fatty changes	20%	34.88%
Centrilobular Necrosis	10%	9.30%
Alcoholic Hepatitis	12.5%	2.33%
Sinusoidal dilatation	5%	6.98%

HPE = histopathological examination

Of the 40 specimens of liver sent for histopathological examination, congestion of the liver was observed in 21 cases (52.5%), followed by 8 cases (20%) showing fatty changes in the liver, 4 cases (10%) revealed centrilobular necrosis and 5 cases (12.5%) of alcoholic hepatitis and 2 cases(5%) of sinusoidal dilation of the liver as depicted in Table 2. All cases (6 cases-100%) which had a survival duration of less than 6 hours showed congestion of the liver. Cases with a survival between 24 hours and 72 hours showed congestion of liver in 4 cases (50%), and 2 cases (25%) each showed fatty changes and alcoholic hepatitis. Cases with a survival beyond 72 hours showed congestion in 11 cases (42.3), fatty change in 6 cases (23%), centrilobular necrosis in 4 cases (15.3%), alcoholic hepatitis in 3 cases (11.5%) and sinusoidal dilation of liver in 2 cases (7.69%) as shown in Table 3.

As shown in Table 4, of the 40 specimens sent, congestion of the lungs was observed in all cases. Associated with congestion, 2 cases showed changes of bronchopneumonia and remaining 38 cases revealed necrosis of epithelium with formation of hyaline membrane. Congestion of the lungs was seen in all cases which had a survival up to 72 hours. Cases with a survival beyond 72 hours showed congestion in 26 cases (100%). Necrosis of epithelium, with formation of hyaline membrane was observed in 38 cases (95%), changes of bronchopneumonia in 2 cases (5%) as described in Table 5.

Of the 40 specimens sent, congestion of both the kidneys was observed in all cases. Associated with congestion, features such as necrosis, degeneration and regeneration of tubular epithelium

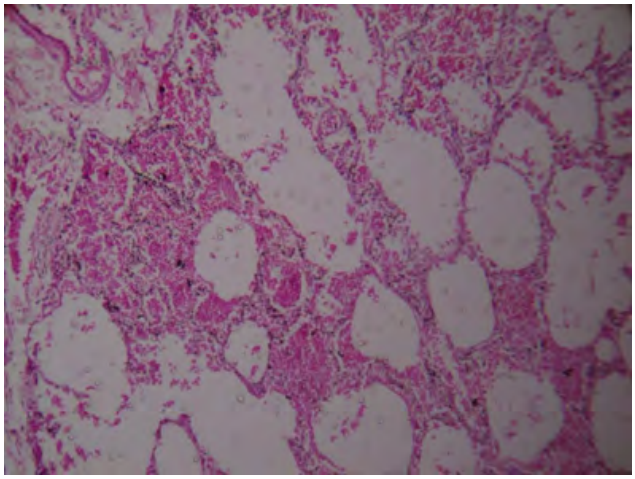

Figure 1: Histopathological slide showing pulmonary congestion in OP Poisoning.

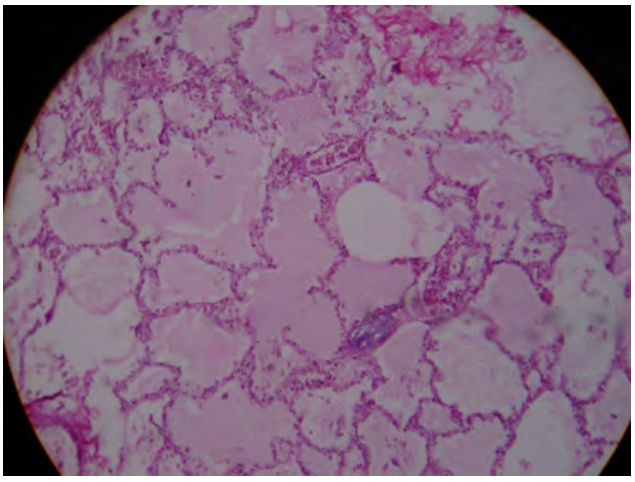

Figure 2: Histopathological slide showing pulmonary edema in Aluminum Phosphide poisoning.

suggestive of acute tubular necrosis were observed in 34 cases and tabulated in Table 6. Congestion of the kidneys was seen in all cases which had a survival up to 24 hours. Congestion associated with necrosis, degeneration and regeneration of tubular epithelium was observed in 8 cases (100%) within duration of survival of 24-72 hours and 26 cases (100%) beyond duration of 72 hours survival as shown in Table 7. Histopathological changes resulting from different poisons have been represented in Figures 1-11.

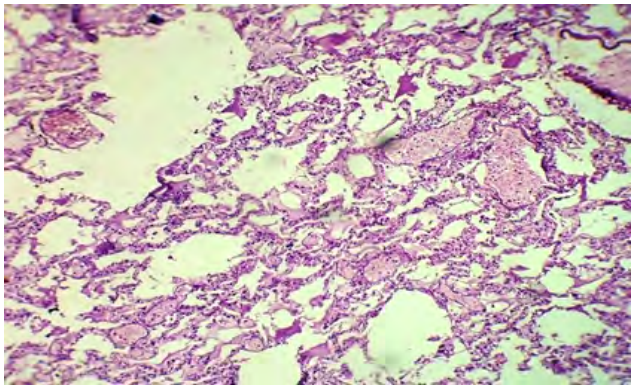

Figure 3: Histopathological slide showing of Diffuse Alveolar Damage in lungs (ARDS) in Carbamate Poisoning.

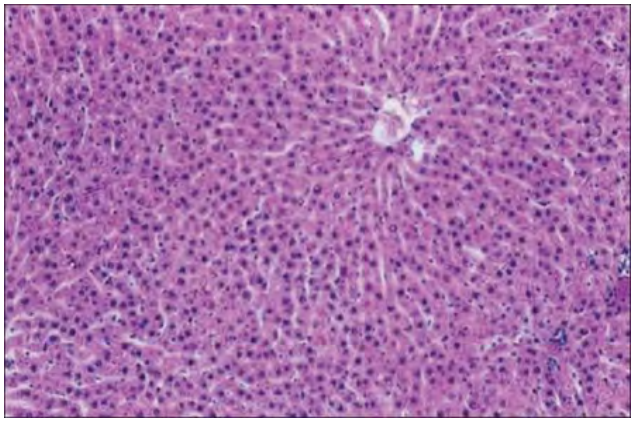

Figure 4: Histopathological slide showing liver congestion in Aluminum phosphide poisoning.

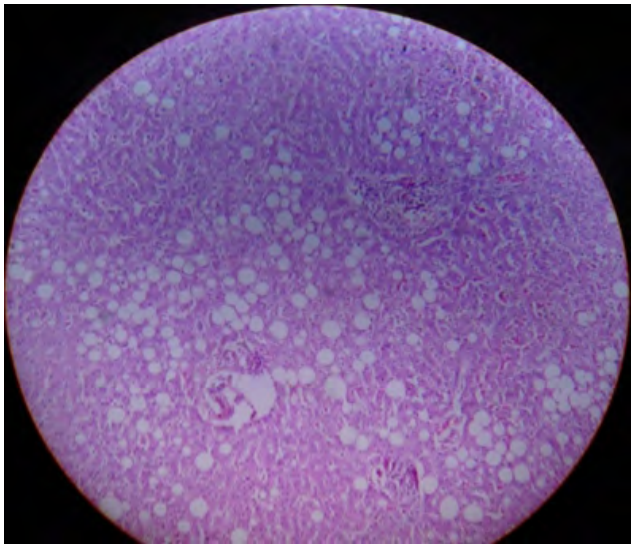

Figure 5: Histopathological slide showing Fatty change in the liver

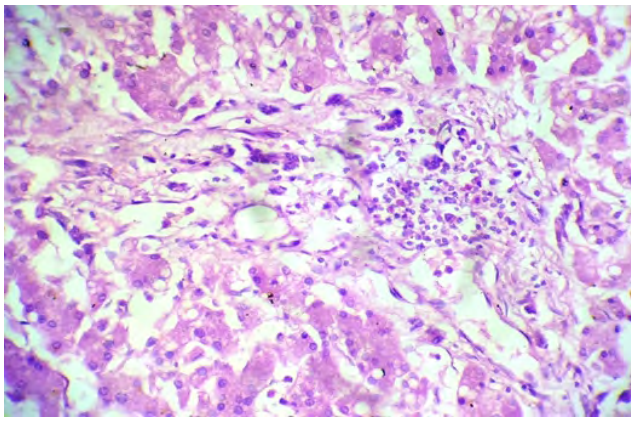

Figure 6: Histopathological slide showing portal inflammation in the liver

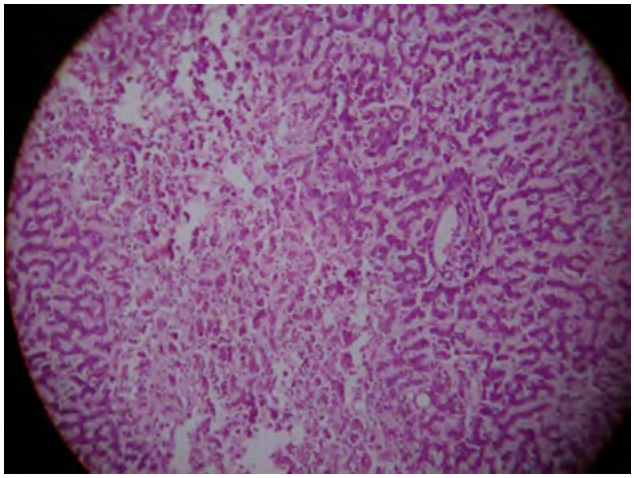

Figure 7: Histopathological slide showing Hepatic necrosis (centrilobular necrosis)

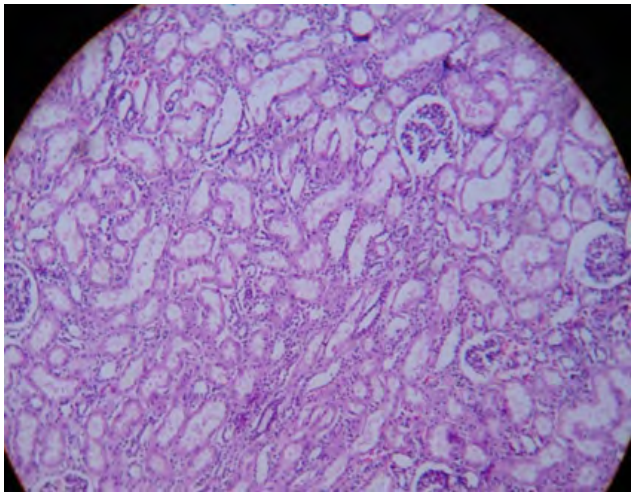

Figure 8: Histopathological slide showing degenerative changes in the kidney

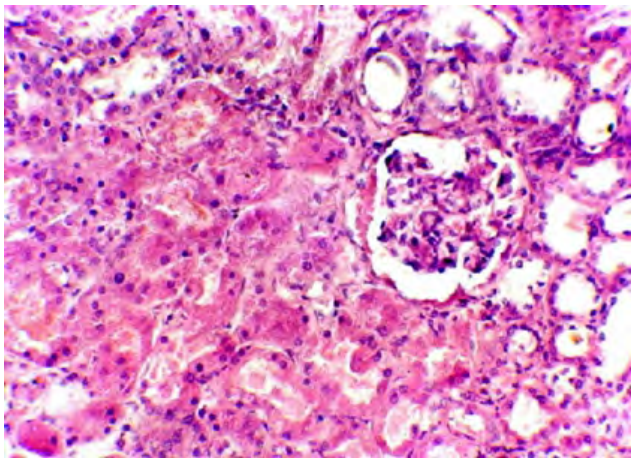

Figure 9: Histopathological slide showing cloudy degeneration in the kidney

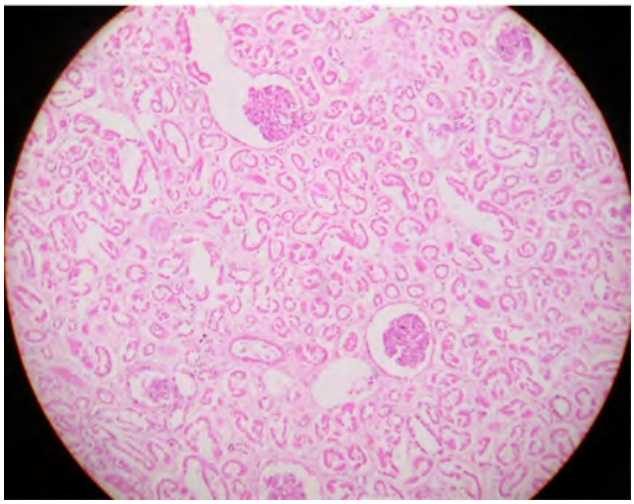

Figure 10: Histopathological slide showing acute tubular necrosis in kidney

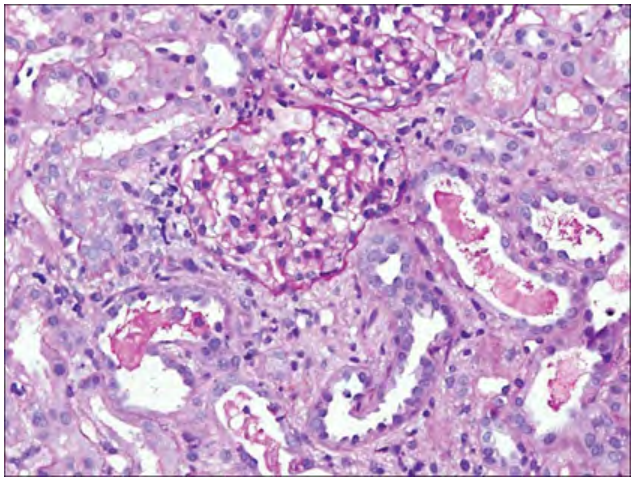

Figure 11: Histopathological slide showing acute tubular necrosis with tubular casts

Discussion

It is interesting to note in the present study that the deliberate ingestion of Organophosphate with suicidal intention is more common in males (65%) compared to that of females (35%) and is consistent with the studies conducted by Rao et al.[4],Gannur et al.[5], Agarawal et al.[6], contrary to the studies conducted by Chataut et al.[2], Paudyal et al.[7] The histopathological findings in the liver in the present study were compared with the findings of the study conducted by Sutay and Tirpude.[8] As depicted in Table 8, congestion of the liver was appreciated in a maximum number of cases in both the studies, 52.5% in the present study and 46.51% in the comparative study, followed by fatty changes in the liver in 20 % of cases in the present study compared to 34.88% centrilobular necrosis of the liver seen in 10% of cases in the present study compared to 9.30% in the comparative study. The microscopic feature least seen in the present study was the sinusoidal dilation of the liver amounting to 5% and the microscopic feature least seen in the comparative study was alcoholic hepatitis of 2.33%. Poison and toxin which are released during metabolism and hemolysis results in organ failure as a consequence of ARDS in lungs, centrilobular necrosis in the liver and acute tubular necrosis of the kidneys. Release of cytokines such as activated macrophages and activated neutrophils results in formation of hyaline membrane resulting in a stiff lung. Bronchopneumonia may be attributed due to aspiration.

Conclusion

In the present study which mainly concentrates on the histopathological findings it is to be noted that all the organs (lungs, liver and kidneys) sent for histopathological examination showed some effects of the poison ingested. The liver is the main organ for detoxification of poison and the kidneys are the main organs of excretion of the poison and this fact was evident from the congestion appreciated in these organs. Features such as ARDS secondary to ingestion was observed in all cases with a survival period beyond 72 hours. Features in the kidneys involving the tubules (degeneration, regeneration and necrosis) were appreciated in almost all cases. Through this study, the confusion among the autopsy surgeon regarding the final cause of death is minimized. A clearer and specific opinion regarding the cause of death can be opined in all cases showing features of ARDS, and death can be opined as respiratory failure resulting from organophosphate poisoning, and in cases showing acute tubular necrosis, the cause of death can be opined as acute renal failure as a result of organophosphate poisoning. Understanding the rapidity of involvement of an organ is beneficial to the treating physician as well thereby helping him reduce the morbidity and hence mortality.

Ethical clearance: A prior approval was obtained from the Institutional Ethics Committee

Conflict of interest: None to declare

Source of funding: None to declare

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