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Original Research Article

Hepatic Histopathology in Fatal Poisoning: An Autopsy-Based Cross-Sectional Study of 65 Cases from a Tertiary Care Centre

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Abstract:

Background: The liver is a major site of xenobiotic metabolism and may show injury in fatal poisoning, but autopsy findings are often non-specific. This study describes hepatic histopathology across commonly encountered poisons and assesses whether hepatic injury may have contributed to death.

Methods: Sixty-five formalin-fixed liver autopsy specimens from documented poisoning deaths were examined over nine months. Sections were stained with H&E, with PAS, reticulin or Masson trichrome when indicated. Congestion, steatosis, bridging fibrosis, necrosis and sinusoidal dilatation were recorded and correlated with age, sex, manner of death and poison group.

Results: Mean age was 34.0±14.5 years; 44 (67.7%) were male and 51 (78.5%) deaths were suicidal. Aluminium phosphide was the commonest individual agent (23, 35.4%); phosphides comprised 28 (43.1%) and organophosphate/pesticide poisoning 20 (30.8%). Congestion was the predominant finding (60, 92.3%), followed by steatosis (18, 27.7%), bridging fibrosis (3, 4.6%), necrosis (3, 4.6%) and sinusoidal dilatation (2, 3.1%). Steatosis was significantly associated with age (p=0.009), but no histological parameter differed significantly by poison group. Hepatic pathology was potentially contributory to death in 3 cases (4.6%).

Conclusion: Hepatic changes are frequent in fatal poisoning but are predominantly non-specific. Congestion appears to reflect terminal circulatory failure, while necrosis was uncommon. Liver histopathology remains valuable for documenting visceral injury, excluding hepatic failure and identifying pre-existing liver disease.

Keywords: Aluminium Phosphide; Autopsy; Forensic Pathology; Hepatic Congestion; Liver; Poisoning; Steatosis.

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Introduction

Acute poisoning remains an important cause of unnatural death in India, with deliberate self-poisoning accounting for a large proportion of fatal cases. The spectrum of agents varies geographically and over time, with agrochemicals, particularly phosphides and organophosphates, commonly encountered. [1-5] The liver is exposed to most ingested toxins through portal circulation and contains abundant xenobiotic-metabolising enzymes. Hepatic injury may therefore result from direct toxicity, metabolic bioactivation or secondary hypoxia and circulatory collapse. [6-8]

Although characteristic hepatic lesions have been described for individual agents, routine medico-legal practice involves a heterogeneous range of poisons. The present study evaluates hepatic histopathological changes in poisoning deaths and their relationship with the implicated agent, demographic variables and possible contribution to death.

Materials and Methods

This observational cross-sectional study was conducted in the histopathology section of the Department of Pathology, Sawai Man Singh Medical College and Attached Hospitals, Jaipur. All adequately preserved liver specimens received during a nine-month period with documented poisoning as the cause of death were included; autolysed or poorly fixed specimens were excluded. Poison type and manner of death were obtained from autopsy requisition and inquest records.

Representative tissue was fixed in 10% neutral buffered formalin, routinely processed and sectioned at 5 µm. H&E was performed in all cases; PAS, reticulin and Masson trichrome were

used when required. Five predefined parameters—congestion, steatosis, bridging fibrosis, necrosis and sinusoidal dilatation—were recorded. Slides were independently reviewed by a consultant pathologist, with discordant interpretations resolved jointly. Necrosis was considered potentially contributory to death; congestion, steatosis and sinusoidal dilatation were regarded as non-specific/reversible changes, while bridging fibrosis indicated pre-existing chronic disease.

Categorical data were expressed as frequency and percentage and continuous data as mean±SD. Chi-square or Fisher exact tests were used as appropriate; $p<0.05$ was considered significant. The study received Institutional Ethics Committee approval.

Results

On gross examination, congestion of the liver was the most frequent abnormality (92.3%), followed by necrosis (64.6%) and gross fatty change (33.8%). Gross examination, however, was insufficient by itself to characterise the injury fully, underscoring the need for systematic microscopic evaluation.

All 65 specimens were adequate for microscopic evaluation. The mean age was 34.0±14.5 years (range 14–75); 26 (40.0%) victims were aged 21–30 years. Males comprised 67.7% and females 30.8%, with one transgender individual. Suicidal poisoning accounted for 78.5% of deaths, accidental poisoning for 18.5%, and manner was undetermined in 3.1%.

Table 1: Demographic profile and manner of death (n=65).

Variable	n (%)
Age ≤20 years	9 (13.8)
Age 21–30 years	26 (40.0)
Age 31–40 years	11 (16.9)
Age 41–50 years	9 (13.8)
Age >50 years	10 (15.4)
Male	44 (67.7)
Female	20 (30.8)
Transgender	1 (1.5)
Suicidal	51 (78.5)
Accidental	12 (18.5)
Undetermined	2 (3.1)

Table 2: Distribution of implicated toxic agents.

Toxic agent	n (%)
Aluminium phosphide	23 (35.4)
Organophosphate compound	16 (24.6)
Zinc phosphide	5 (7.7)
Unspecified pesticide	4 (6.2)
Methanol	4 (6.2)
Paracetamol/drug overdose	4 (6.2)
Copper sulphate	3 (4.6)
Corrosive acid	3 (4.6)
Unknown poison	3 (4.6)
Total	65 (100.0)

Phosphides accounted for 43.1% of cases, organophosphate/other pesticides for 30.8%, and other agents for 26.2%. The distribution of toxic agents was significantly non-uniform ($p<0.001$). Phosphide and organophosphate deaths were concentrated in the 21–30-year age group. Sex and manner of death did not differ significantly among poison groups.

Hepatic Histopathology

Table 3: Microscopic hepatic findings (n=65). Multiple findings could occur in one case.

Finding	n (%)
Congestion	60 (92.3)
Steatosis	18 (27.7)
Bridging fibrosis	3 (4.6)
Necrosis	3 (4.6)
Sinusoidal dilatation	2 (3.1)
No abnormality	1 (1.5)

Congestion was the dominant lesion and occurred in 92.3% of specimens. Steatosis occurred in 27.7%; bridging fibrosis and necrosis in 4.6% each; sinusoidal dilatation in 3.1%. Overall, 86 lesions were recorded, with a mean lesion burden of 1.3±0.6 per case. Steatosis was significantly associated with age (p=0.009), being absent below 20 years and highest in the 31–40-year group (63.6%). No histopathological parameter differed significantly by sex or poison group.

Necrosis was absent in all 28 phosphide cases and occurred in one organophosphate and two other-agent cases. On the prespecified criterion, potentially contributory hepatic pathology was identified in 3/65 cases (4.6%), all outside the phosphide group. Representative microscopic findings included sinusoidal dilatation, congestion, macrovesicular steatosis, bridging fibrosis and hepatocyte necrosis.

Discussion

This study demonstrates that hepatic abnormality is common in fatal poisoning but is rarely specific for the causative agent. The demographic profile was characterised by young adults, male predominance and a high proportion of suicidal deaths, consistent with previous Indian autopsy series. [1,4,5] Aluminium phosphide was the commonest individual agent (35.4%), although its proportion was lower than that reported in some northern Indian series, highlighting regional variation in poisoning patterns. [5,17,18]

The principal finding was hepatic congestion in 92.3% of cases, with similar frequencies across phosphide, organophosphate/pesticide and other-agent groups. Such widespread occurrence argues against congestion being a toxin-specific lesion. Previous studies of aluminium phosphide poisoning have similarly associated visceral congestion with severe hypotension, tissue hypoxia and terminal circulatory collapse. [19-21] In forensic reporting, congestion should therefore be interpreted as a non-specific circulatory/agonal change rather than proof of direct toxic hepatic injury.

Steatosis was the second most frequent finding (27.7%) and was significantly related to age rather than poison group. Its highest prevalence in the 31–40-year group and absence below 20 years suggest that much of the fatty change represents background metabolic or pre-existing liver pathology rather than a direct toxic effect. This interpretation is supported by autopsy studies showing substantial prevalence of steatosis in unselected deaths. [22]

Necrosis was uncommon (4.6%) and was not observed in phosphide poisoning. The three necrotic cases—all outside the phosphide group—were the cases classified as having potentially contributory hepatic pathology. This contrasts with the rapid haemodynamic collapse typical of severe aluminium phosphide poisoning and with agents such as paracetamol, in which hepatocellular necrosis is a characteristic toxic lesion. [12,19,23]

The fine isomorphic cytoplasmic vacuolation reported in some phosphine-poisoning series was not specifically recorded in this study. Its subtle nature, variable fixation and post-mortem interval may contribute to under-recognition. Because the study was designed around five predefined parameters, this finding requires dedicated evaluation in future studies.

Forensic Significance: Although only 4.6% of cases showed hepatic pathology potentially contributing to death, routine liver examination remains useful. Histopathology can document visceral injury, identify pre-existing chronic liver disease, and provide objective evidence when toxicological results are pending. Importantly, non-specific lesions should not be over-interpreted as agent-specific toxic injury.

Strengths and Limitations: Strengths include systematic assessment of consecutive specimens using predefined parameters, special stains when indicated, independent slide review and a prespecified criterion for contributory hepatic pathology. Limitations include the single-centre design, modest sample size, dependence on autopsy/inquest documentation for poison identification, incomplete quantitative toxicological confirmation, fragmentary liver submission, unrecorded post-mortem intervals and the restricted histological panel. These factors limit generalisability and may have reduced detection of subtle or focal lesions.

Conclusion

Hepatic involvement was frequent in fatal poisoning but was dominated by non-specific congestion, which occurred irrespective of poison group and is best interpreted as a marker of terminal circulatory failure. Steatosis was associated with age rather than the implicated toxin. Necrosis was uncommon and was the principal lesion considered potentially contributory to death. Liver histopathology therefore seldom identifies the poison by itself, but remains valuable in medico-legal practice for documenting injury, excluding hepatic failure and recognising pre-existing disease. Larger multicentre studies incorporating quantitative toxicology, post-mortem intervals and a broader histological panel are warranted.

Declarations

Ethics Approval: Approved by the Institutional Ethics Committee of Sawai Man Singh Medical

College, Jaipur. Conflicts of interest: None declared.

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Data availability: Anonymised data are available from the corresponding author on reasonable request.

Author contributions: SS conceived the study, examined specimens, collected/analysed data and drafted the manuscript; JM supervised histopathological assessment and critically revised the manuscript; VK provided departmental oversight and reviewed the final draft. All authors approved the final version.

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