

# Acute Methanol Toxicity: Correlation with Autopsy Findings, A Descriptive Study

Autopsy Findings  
in Acute  
Methanol  
Toxicity

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## ABSTRACT

**Objective:** This paper presents postmortem and histological data in order to clarify the inner progression of methanol intoxication. The research will also examine the histological, biochemical, and clinical changes brought on by methanol poisoning.

**Study Design:** Descriptive cross-sectional investigation.

**Place and Duration of Study:** This study was conducted at the Department of Forensic Medicine and Toxicology, DHQ, Rawalpindi, Rawalpindi Medical University, Rawalpindi from January 2022 to October 2022.

**Materials and Methods:** Four persons were brought in dead; four of them passed away shortly after being admitted (early deaths), five of them passed away between 10 and 45 days after being admitted to the hospital (late deaths). Each of the 13 victims had a medico-legal autopsy. Gross exterior and internal results were recorded, and routine blood and viscera conservation & transfer to the lab for methyl alcohol analysis were also carried out. From the optic chiasma, a section of the nerve that supplies vision was removed for histological analysis. Information was gathered using old records.

**Results:** Their age ranged from 20 to 60 years old, and 70% (n = 10) of all fatalities were between 25 and 55 years old. The average methyl alcohol concentration was 107.12 mg/dl and 236.8 mg/dl, respectively, among hospital fatalities and brought deceased. Vomiting and stomach discomfort were the most frequently reported symptoms (n = 8), followed by poor eyesight (77%), and indicators of respiratory insufficiency (52%). Hospitalization deaths had average bicarbonate & pH values of 7.29 and 7.72mmol/l, correspondingly. An autopsy revealed that each person had hypoxia. The obstruction affected internal organs. In the early instances, serious acidosis of the metabolism that led to respiratory failure was the cause of death. As a consequence of septicemic shock, brain and pulmonary edoema led to late deaths. Cerebral edoema and intracerebral haemorrhage were seen in the patient with the longest survival duration. Another internal finding in late deaths was the presence of cirrhotic liver together with symptoms of renal failure. The optic nerve's histopathology showed modest edematous changes rather than demyelination or axonal necrosis.

**Conclusion:** Poisoning by methanol. The many organs and organ systems of the body have a number of detrimental effects. Many lives may be saved with an early diagnosis and treatment. Organ-directed, accurate autopsies may help autopsy surgeons make diagnoses and, as a result, can help the legal system bring perpetrators to justice.

**Key Words:** Acute Methanol Toxicity, Correlation, Autopsy Findings

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## INTRODUCTION

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Methanol is a highly hazardous chemical found in chemicals, antifreeze, and fuel components. Orally taken formic acid suppresses cytochrome oxidase within the mitochondrial electron transport chain because it is easily metabolized from methanol to formaldehyde and other compounds. This defect causes metabolic acidosis and has an impact on the optic nerve and other elements of the central nervous system<sup>(1, 2)</sup>. Methanol is a clear, colorless liquid that smells and tastes like alcohol. It is commonly utilized as a raw ingredient for industrial solvents, antifreeze manufacture, and ethyl alcohol denaturation. Oral intake, both accidental and suicide, has serious visual and neurological consequences. The clinical outcome spans a wide range, from visual neuropathy to death<sup>(3,4)</sup>. The chemical breakdown of methanol is

mostly controlled by the liver.. Aldehyde dehydrogenase then transforms formaldehyde into formic acid when methyl alcohol is first oxidized to generate it<sup>(5)</sup>. The majority of the negative side effects of intoxication are caused by these metabolites. Due to a delay in the formation of these metabolites, there is typically a delay between methanol consumption and the onset of symptoms. Due to this latency, the diagnosis of intoxication may go unrecognized.

Among the hazardous consequences that are often seen are ophthalmological problems (6), blindness, optical nerve damage, brain edoema, acute renal failure, and severe metabolic changes. A substantial corpus of case studies and research supports the early diagnosis and therapeutic treatment method (7, 8). However, few investigations have compared the medical & chemical profiles of instances of poisoning with methanol at the time of death with the outcomes. In order to comprehend the internal development of methanol intoxication, this paper discusses the postmortem and histological findings. The study investigates the histological, biochemical, and clinical modifications brought on by methanol overdose.

## MATERIALS AND METHODS

In this cross-sectional study carried out at Department of Forensic Medicine and Toxicology, DHQ, Rawalpindi, Rawalpindi Medical University, Rawalpindi. Four people were brought in dead; five more perished between 10 and 45 days after being admitted to the hospital, and four of them passed away shortly after being admitted to the hospital (early deaths). The fatalities were separated between brought fatalities (n = 6) and hospital deaths (n = 10), or those who were brought in alive but passed away while receiving care. All 13 dead were subjected to medical-legal autopsies. Gross exterior and internal anomalies were seen, and blood samples and routinely stored viscera were sent to a lab for methyl alcohol analysis. A portion of the optic nerve was taken from the optic chiasma for histological analysis.

Reviewing hospital records in order to compile essential information on the occurrence history, clinical examination, biochemical profile, treatment, and result was part of the retrospective data gathering process. To obtain pertinent information, interviews with the deceased's relatives who were brought dead were also conducted.

## RESULTS

A serious public health issue is methanol poisoning. Histopathological analyses performed during autopsies may show that methanol has a negative impact on a number of organs. Their age ranged from 20 to 60 years old, and 70% (n = 10) of all fatalities were between 25 and 55 years old. The average methyl alcohol concentration was 107.12 mg/dl and 236.8 mg/dl,

respectively, among hospital fatalities and brought deceased.

Vomiting and stomach discomfort were the most frequently reported symptoms (n = 8), followed by poor eyesight (77%), and indicators of respiratory insufficiency (52%). The average pH and bicarbonate levels in hospital fatalities were 7.72 mmol/l and 7.29 mmol/l, respectively. All of the patients' hypoxia was verified by the autopsy. Internal organs were obstructed. The cause of death in the early cases was severe metabolic acidosis that resulted in respiratory failure. As a consequence of septicemic shock, brain and pulmonary edoema led to late deaths. Cerebral edoema and intracerebral haemorrhage were seen in the patient with the longest survival duration. Another internal finding in late deaths was the presence of cirrhotic liver together with symptoms of renal failure. The optic nerve's histopathology showed modest edematous changes rather than demyelination or axonal necrosis.

**Table No. 1: Profile of the cases of methanol poisoning**

Parameters	Values
Methanol poisoning cases overall	100
Brought dead	04
Total deaths	04
Death in hospital	05
Average age (min-max) of all deaths in a year	34.0
The age group most influenced in years	25-55, 70% (N = 10)
Sex (male)	100%
Mean postmortem interval	14.6 HOURS

**Table No. 2: Clinical descriptions of cases of methanol poisoning**

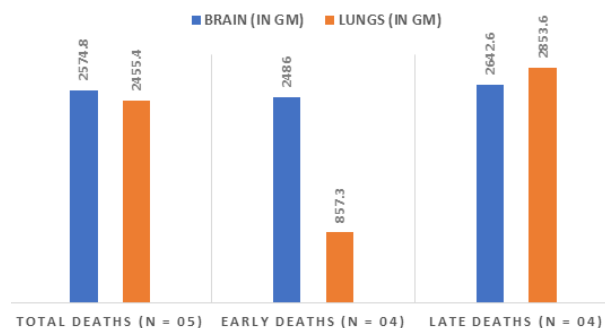
Clinical Indicators	Hospital Fatalities (N = 8)	Immediate Deaths (N = 4)
Average Methanol Concentration MG/DL)	107.12	236.8
Underwent Hemodialysis	100%	Not Applicable
Initial Clinical Presentations		
Neurological Symptoms	100%	Not Evaluated
Ophthalmic Manifestations	77% (N = 8)	Not Assessed
Gastrointestinal Signs	39.7% (N = 5)	Not Observed
Respiratory Issues	52% (N = 6)	Not Examined
Mean Arterial pH	7.72	Not Available
Average Bicarbonate Level (MMOL/L)	7.29	Not Recorded

All individuals who died in hospitals were admitted in a coma. Their most frequent complaint was hazy vision and sudden vision loss, which was reported by 77% (n = 8), followed by nausea and excruciating stomach pain, and 52% (n = 6) also had symptoms of respiratory insufficiency. The mean pH and bicarbonate levels of hospital fatalities were 7.72 mmol/l and 7.29 mmol/l, respectively. Patients who died away in hospitals revealed evidence of acute renal failure during biochemical testing.

The average postmortem period was 14.6 hours, and all of the cases' autopsies showed evidence of hypoxia. Internal organs were congested, and the average weight of the brain and lungs was increased overall in deaths. Early deaths were caused by respiratory failure brought on by severe metabolic acidosis. Late deaths were caused by cerebral and pulmonary edema as a result of septicemic shock. In a case with the longest survival time, there was cerebral edema and intracerebral hemorrhage affecting the intrathalamic area and bilateral basal ganglia, both of which were encircled by decaying tissues. A cirrhotic liver and indications of renal failure were additional internal findings in late deaths. In the histology of the optic nerve, there was no evidence of demyelination or axonal necrosis, however there were slight edematous alterations.

**Table No. 3: Average weight of lungs and brain**

Weight of the brain and lungs among	Brain (in gm)	Lungs (in gm)
Total deaths (n = 05)	2574.8	2455.4
Early deaths (n = 04)	2486	857.3
Late deaths (n = 04)	2642.6	2853.6



**Figure No. 1: Average lungs and brain weight.**

## DISCUSSION

The medical & postmortem characteristics of the deceased are compared and contrasted in this study to better understand the rapid effects of methanol intoxication. Metabolic acidosis, which caused respiratory failure, was the main cause of the early mortality. The main factor in late mortality was sepsis, which caused multi-organ failure. The oxidation of methyl alcohol produces formic acid, which leads to

metabolic acidosis. Mortality and metabolic acidosis are associated.

The victims in this study were 34.01 years old on average. The most affected were low socioeconomic males between the ages of 25 and 55. These findings are consistent with other research<sup>(9, 10)</sup> findings that middle-aged men are particularly vulnerable. Methanol levels in hospital and non-hospital fatalities were, respectively, 107.12 mg/dl and 236.6 mg/dl on average. However, many studies have shown that the lethal methanol level may vary. The lethal blood level for methanol was shown to vary from 60 to 200 mg/dl in other trials, with the lowest and highest amounts being 76 and 337 mg/dl, respectively<sup>(11)</sup>. The same quantity of methanol may have different effects on various people<sup>(12)</sup>. The same quantity of methanol may have different effects on different persons<sup>(13)</sup>. This can be the result of a number of factors, including demographic differences, coexisting illnesses, tolerance levels, and metabolic rates.

Patients with methanol intoxication often report neurological issues, gastrointestinal issues<sup>(14)</sup>, and visual abnormalities<sup>(15)</sup>. In the current investigation, a coma was present in every hospital death. The accompanying people's histories revealed that ocular symptoms predominated (77%; n = 08), followed by respiratory symptoms (52%; n = 6) and gastrointestinal symptoms (38.6%; n = 5). The bulk of hospital deaths were caused by comas or breathing difficulties, according to recent study<sup>(16)</sup>. Common neurological symptoms reported in clinical settings include headache, vertigo, altered sensorium, and coma<sup>(17, 18)</sup>. The most often injured organs that cause mortality, according to research, are the kidney and the brain. Cerebral edoema, cerebral congestion, intracerebral haemorrhages, and degenerative necrosis are the known findings in the central nervous system. Pulmonary edoema, superficial pulmonary haemorrhages, and fatty liver changes are additional usual findings. Patchy tubular degeneration and necrosis are possible symptoms of renal damage.

The results of our investigation meet the criteria listed above. Despite being present in all cases, cerebral and pulmonary edoema was more pronounced in the subsequent deaths, as seen by the growing weight of the brain and lungs. Elderly victims exhibited fatty changes, while the longest-surviving victim had an intracerebral haemorrhage affecting the basal ganglia and thalamus. The optic nerve from an optic chiasm had moderate edoema but no demyelination or axonal necrosis, according to histopathology. However, several studies have shown significant demyelination along with intra-axonal enlargement and organelle death<sup>(19)</sup>.

Our investigation was severely hampered by the very small sample size. It would have been easier to explain the victims' different clinical traits if we had

simultaneously measured the amounts of ethanol, methanol, and its metabolites.

## CONCLUSION

In many areas of India, poisoning with methanol poses a serious threat to public health. Histopathological analyses, which are part of autopsies, may show how destructive methanol is to different organs while also helping the legal system do the victim's killer justice. The ocular, cerebral, pulmonary, and renal systems are typically impacted by methanol poisoning. Look for indications of brain, pulmonary, and renal impairment both at autopsy and in the emergency room. It's also possible to find intracerebral haemorrhages. The general population must be made aware of the risks associated with ingesting tainted methanol, and the government must enact strict regulations to control the production of illicit spirits.

### Author's Contribution:

Concept & Design of Study: Sheeba Shabbir  
 Drafting: Muhammad Shafay, Tasneem Murad  
 Data Analysis: Filza Ali, Nazia Yasmin  
 Revisiting Critically: Sheeba Shabbir, Muhammad Shafay  
 Final Approval of version: Sheeba Shabbir

**Conflict of Interest:** The study has no conflict of interest to declare by any author.

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