ISSN 2394–806X (Print), ISSN 2454-5139 (Electronic)
IJHRMLP, Vol: 06 No: 01, January, 2020
Printed in India

CASE REPORT

Fatal formalin poisoning - a case report on forensic histopathology

Das Abhishek[1], Ejaz Ambreen[2], Das Nandini[3], Sukul Biswajit[4]

Received on October 20, 2019; editorial approval on November 25, 2019

ABSTRACT

Formalin poisoning, mostly accidental, if not suicidal, is relatively common in those areas where the chemical is used or easily available like textile factory, rubber factory, printing etc. Strong irritation and offensive, pungent odour contributes to its rarity. Formalin poisoning affects all the systems as it is easily absorbed through respiratory and gastrointestinal tract and spreads in whole body through blood causing constriction in chest, palpitation, pain abdomen and gastrointestinal irritation, urinary abnormality, liver damage and clotting factors abnormality. This type of poisoning is rare and unusual. It's associated with high mortality rate, so proper elicitation of history, identification of the offending material and postmortem histopathology are essential. A 68 year old male was admitted with history of formalin poisoning after 7 hours and expired 40 mins post admission Evident findings were prominent on autopsy followed by histopathology. The present investigation is an attempt to highlight the clinical picture along with autopsy histopathology in details of formalin poisoning. Such studies are essential in developing appropriate preventive strategies.

Keywords: *Submucosal petechial haemorrhages; acute tubular necrosis, steatosis; autopsy histopathology.*

INTRODUCTION

Formalin is a colourless liquid organic compound which is used both in healthcare industry and other industries. "All crystal clear liquid is not water" so goes the saying and it might lead to death. Accidental & suicidal formalin poisoning, usually accidental if not suicidal, is relatively common in those areas where this chemical is easily available, like in a rubber factory, printing & textile factories etc. Formalin is aqueous solution of formaldehyde, with about 37% to 40% of formaldehyde in it.[1] It is colourless water like liquid which on oxidation produces formic acid which is also colourless with pungent, penetrating odour.[2] Relatively slow metabolism of formic acid in humans lead to its accumulation leading to metabolic acidosis. Formic acid is also an inhibitor of mitochondrial cytochrome oxidase to produce histotoxic hypoxia so that a significant part of the acid load results from hypoxic metabolism.[3] It is irritating, corrosive, toxic & absorbed from all surfaces of body. Moreover it is a protoplasmic poison causing tissue fixation, coagulation necrosis, and protein precipitation.[4] Few cases of fatal formalin have been reported in literature, but none depicts the combined and comprehensive array including clinical features, external and internal autopsy findings and histopathological profile with supporting figures together which has been attempted here.

CASE HISTORY

A 68 year old male was admitted with history of formalin poisoning 7 hrs 35 mins back. He was referred from local hospital after initial management. On admission patient had

Address for correspondence:
[1]Assistant Professor
Mobile: +918902640596
Email: abhishek.das.forensic@gmail.com
[2] Post Graduate Trainee
Department of FSM, Medical College, Kolkata
[3] Demonstrator (**Corresponding author**)
Mobile: **+**919681992794
Email: nandini1004@gmail.com
Department of Pathology
[4] Professor & Head, FSM, Medical College, Kolkata.

Cite this article as: *Das Abhishek, Ejaz Ambreen, Das Nandini, Sukul Biswajit. Fatal formalin poisoning – a case report on forensic histopathology. Int J Health Res Medico Leg Prae 2020 January;6(1):71-74. DOI 10.31741/ijhrmlp.v6.i1.2020.15*

hypotension, tachycardia. He expired 40 mins after admission. Medicolegal autopsy was performed.

POST MORTEM FINDINGS

On external finding, conjunctiva was congested. There was greyish white discolouration in the inner aspect of both lips and tongue (**Figure 1**). External examination was unremarkable without any external injury. On opening the abdominal cavity, yellowish transparent peritoneal fluid was noted in small amount, intestinal walls appeared congested with evidence of patchy haemorrhages at places. Small intestine, on dissection, appeared to be thinned out, had lost its rugosity and was shrunken (**Figure 2**). On opening the oesophageal lumen, mucosal longitudinal streaky haemorrhages were seen. Mucous membrane of stomach was congested with multiple submucosal petechial haemorrhages at places. White streaks were seen on internal surface at places with blunt gastric rugae. Stomach contained small amount of partly digested food without any appreciable odour. Kidneys showed evidence of haemorrhages on its surface. On sectioning, poor cortico-medullary differentiation and haemorrhagic spots were seen. Liver showed patchy subcapsular haemorrhages at places.

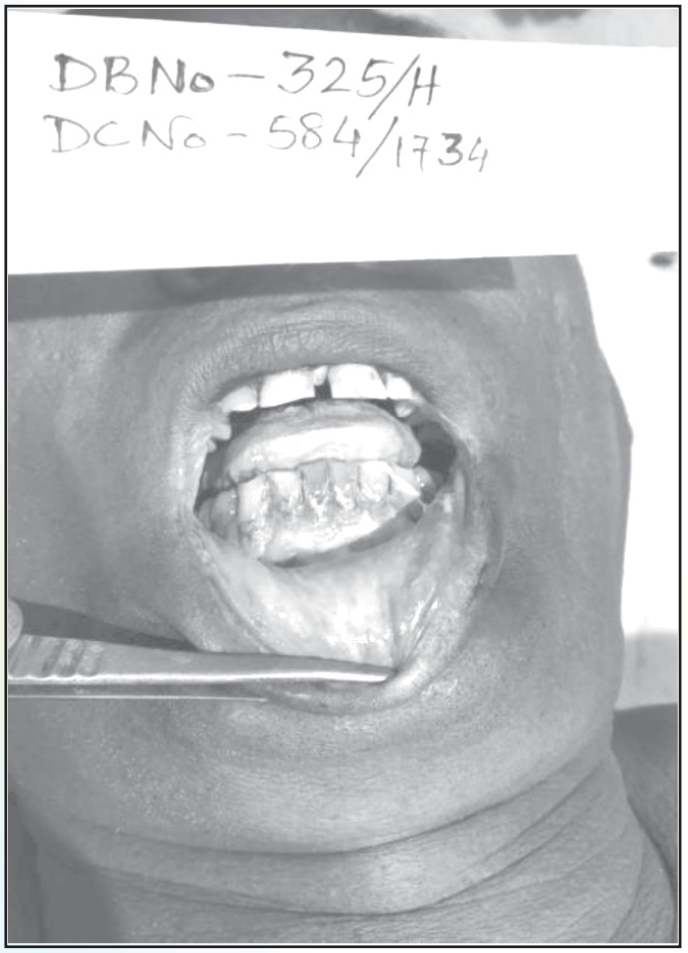

Figure 1 Greyish white discolouration in the inner aspect of both lips and tongue

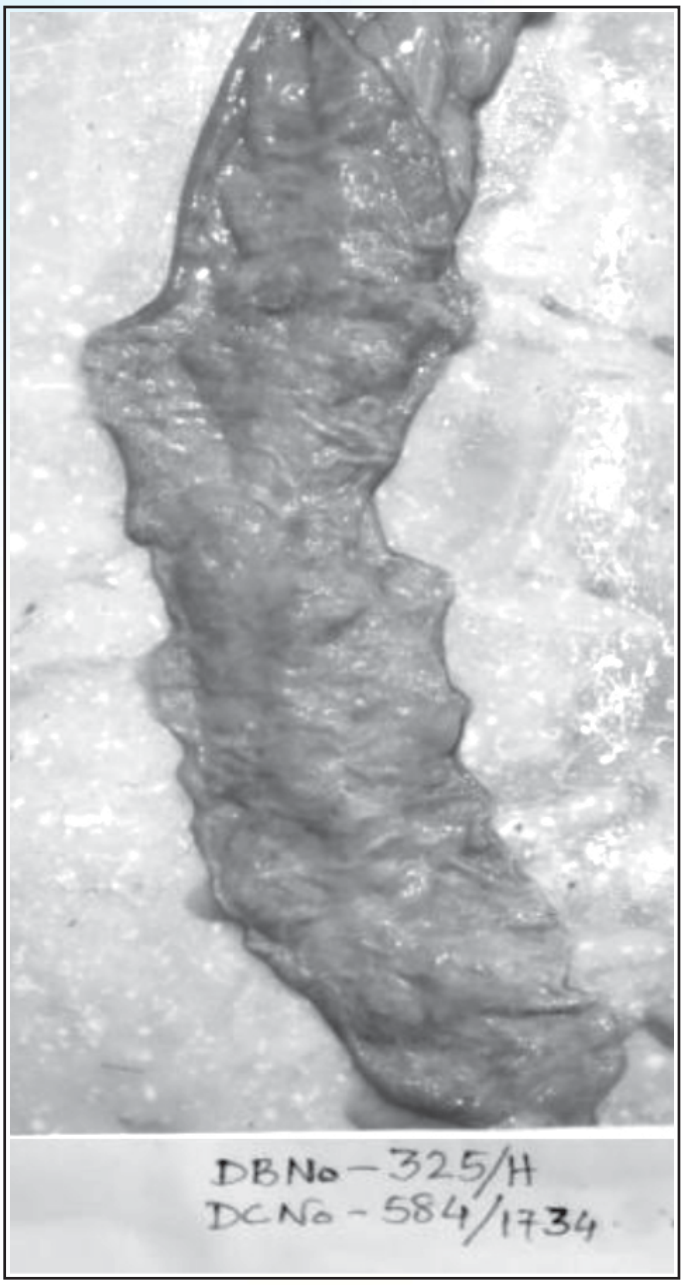

Figure 2 Thinned out, shrunken small intestine with loss of rugosity

HISTOPATHOLOGY FINDINGS

Stomach showed mucosal denudation & slough (altered exudates) at places along with polymorphonuclear infiltration (**Figure 3**). Gross congestion & haemorrhage at places seen, gastric glands are fixed and prominent with markedly oedematous lamina propria. Small intestine showed congestion and haemorrhage at places oedematous lamina propria. Kidney showed glomerular congestion, interstitial edema; interstitial haemorrhage with typical features of acute tubular necrosis (**Figure 4**). Features of steatosis with evidence of droplets, both microvesicular & macrovesicular was seen in liver (**Figure 5**). Increased infiltration of subcapsular lymphocytes was detected in liver.

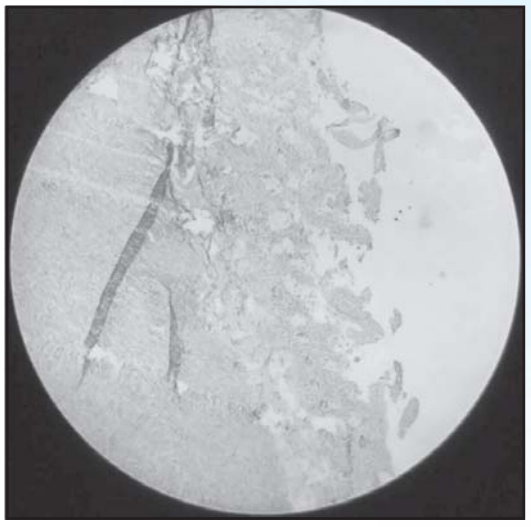

Figure 3 Mucosal denudation and slough with PMN infiltration, congestion and haemorrhage in stomach

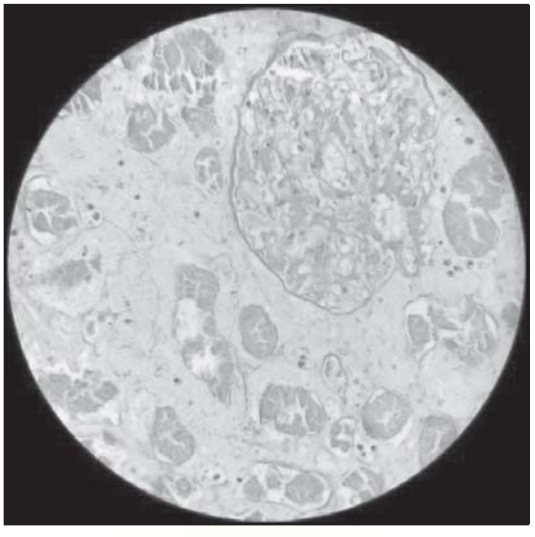

Figure 4 Typical features of acute tubular necrosis in kidney

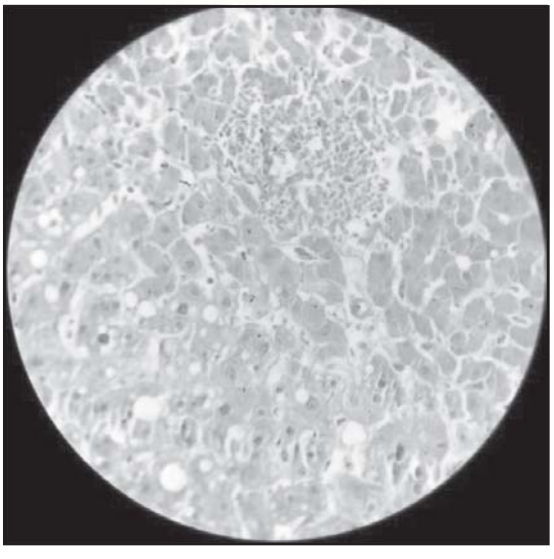

Figure 5 Steatosis with microvesicular and macrovesicular droplets in liver

DISCUSSION

Few cases of fatal formalin have been reported in literature, but none depicts the combined and comprehensive array including clinical, autopsy and histopathological profile together which has been attempted here. Ingestion of as little as 30 ml (2 tablespoon quantity) of 37% formaldehyde solution (formalin) has been reported to cause death in an adult, preceded by gastrointestinal absorption leading to severe corrosive damage to stomach and small intestine, circulatory collapse and kidney damage.[4] Another case report depicts that the ingestion of formalin can lead to chemical peritonitis even without perforation.[5] Case report from Karnataka found that renal failure is a frequent complication in this poisoning due to metabolic acidosis. Kidneys may also reveal microscopic evidence of acute tubular necrosis, which is consistent with authors' findings. Formaldehyde is a corrosive material that can make the skin & mucous membrane appear whitened.[6] This was also seen in this case. A case report in Netherlands stated that systemic effects that can occur are shock, unconsciousness, convulsions and acute respiratory failure. Formalin poisoning frequently causes multiorgan failure.[7] Another case report from India revealed stomach appearing like tea-pot with formalin smell and brownish black appearance of mucosa. Clinical effects of acute poisoning include hypotension and cardiovascular collapse; upper respiratory tract irritation, coughing, brownish black appearance bronchitis, pulmonary oedema, or pneumonia, ARDS (adult respiratory distress syndrome); lethargy and coma; nausea,vomiting, and severe abdominal pain; corrosive gastritis, oedema, ulceration and even perforation of the oesophagus.[8] The stomach suffers the most severe damage in such cases because formalin is in contact with the gastric mucosa longer than other parts of the gastrointestinal tract. The phenomenon of perimortal fixation is a useful indication for the forensic pathologist and should direct the suspicion to oral poisoning.[9] The introduction of formalin into the stomach is followed by the production of a gastritis which varies greatly in character. The duodenum and upper jejunum may also be involved in the inflammatory process. Formalin in whatever way introduced into the body is absorbed, and is then capable of producing lesions in the parenchymatous organs.[10] Extent of formalin induced corrosive injury varies greatly upon duration of contact with mucosal surface. Oesophageal burn is rare due to prompt passage along through oesophagus, though in present cases streaky haemorrhages seen longitudinally along oesophagus.[3] As whole esophagus, stomach, small intestine, kidney and liver are the target organs.[2] Cause of death may be varied as reported in various clinical reports which includes acute tubular necrosis,[3] metabolic acidosis,[4] histotoxic hypoxia[6] acute respiratory failure[7] circulatory collapse.[8] Steatosis is not a feature of acute formalin poisoning, rather of chronic formalin exposure, though this case showed the said feature. Upon questioning the co-workers and fellow colleagues of the subject, they admitted that formalin was usually kept in common bottles (same as that of drinking water) and very often kept open. So

the vapours of formalin used to mix in the air to be inhaled by the workers leading to a chronic exposure. This was the circumstance of the poisoning that the subject mistakenly took formalin instead of drinking water. Toxicological analysis from forensic science laboratory also confirmed the presence of aldehyde in the samples of blood and viscera.

CONCLUSION

Treatment of these patients must be started promptly. Hypotension should be treated with fluids & vasopressors, metabolic acidosis with sodium bicarbonate judiciously. Forensic experts should be alert in such cases & proper autopsy protocol should be followed. Meticulous dissection & proper preservation of viscera for chemical analysis & histopathology is essential in such cases. Ingestion of formalin in significant amount usually has fatal consequences.

Conflict of interest: None declared.
Ethical clearance: Taken.
Source of funding: None declared.
Author disclosure: (1) The article is original with the author(s) and does not infringe any copyright or violate any other right of any third party. (2) The article has not been published (whole or in part) elsewhere and is not being considered for publication elsewhere in any form, except as provided herein. (3) All author(s) have contributed sufficiently in the article to take public responsibility for it and (4) all author(s) have reviewed the final version of the above manuscript and approved it for publication.

REFERENCES

1. Aggarwal A. Textbook of Forensic Medicine and Toxicology. 1st ed. Avichal Publishing Company; 2016;p.750.
2. Pillay VV. Modern medical toxicology. 4th ed. Jaypee; 2013;p.352-4.
3. Pandey CK, Agarwal A, Baronia A, Singh N. Toxicity of ingested formalin and its management. Human and experimental toxicology 2000;19:360-6.
4. Raut SM, Haridas SV, Kachare RV, Pawar VG, Dode PS. Fatal formalin poisoning: a rare case report. IJFMTS Oct-Dec 2018;3(4):100-2.
5. Yanagawa Y, Kaneko N, Hatanaka K, Sakamoto T, Okada Y, Yoshimitu S. A case of attempted suicide from the ingestion of formalin. Clin Toxical 2007; 45(1):72-6.
6. Chakraborty S, Veeresh MR. Formalin, a rare suicidal poison- a case report. IOSR- JDMS April 2015; 14(4)24-6.
7. Vos H, Lunistra M, Pauw R. Survival of a formalin intoxication: a case report. Netherlands J of critical care Jan-May 2017;25(4):133-6.
8. Gadhari RK, Pathak AG, Chaudhari KM, Devraj NA. Fatal self poisoning with formalin: a case report. J of Medicolegal Association of Maharashtra Jul-Dec 2014;23:2.
9. Hungund Chandrakanth, Mohanram Arun, Pai Varun, Rani Smitha. Fatal formalin poisoning: a case report. Iranian J of Toxicology 2011;5(1):468-9.
10. Shetty P, Bakkannavar SM, Goud PK. Formalin poisoning - a case report. Poster Presentation. Asia Pacific Association of Medical Toxicology. 13th International Scientific Congress- Shenyang. pp.017.