

Oral Cavity: An insight to forensic diagnosis

Sneha Sethi¹, Poonam Goel², Sonam Bhalla³

¹Senior Lecturer, Sudha Rustagi College of Dental Sciences and Research, Faridabad, Haryana, India

²Senior Lecturer, Seema Dental College, Rishikesh, Uttarakhand, India

³M.D.S Oral Pathology, working at Sham polyclinic, Trunk market, Rajpura town, distt Patiala, Punjab, India

ABSTRACT

Forensics is a branch of medicine that deals with the legal implications of healthcare, determines and interprets causes of disease, injury and death. Various causes of death are known to show different features in the entire human body including oral cavity. These features of the oral cavity can be used as a remote identity to assess the cause of death. The oral cavity can be considered as a region with tremendous potential especially in regard to coming to a final forensic diagnosis, but this aspect of forensic medicine is relatively undiscovered and overlooked. This article revolves around this concept and reviews the different manifestations in the oral cavity observed post mortem.

Keywords: diagnosis, forensics, oral cavity, postmortem.

Introduction

Law has not tried to define death. By death, Black's law dictionary means "cessation of life" or "ceasing to exist." Medico-legally, death is defined as permanent and irreversible cessation of functions of the three interlinked vital systems of the body which are called the tripod of life, namely the nervous, circulatory and respiratory systems. A doctor has to prevent death of a person, pronounce a person dead when his terminates, ascertain the time and cause of death.[1] Despite medical and technical advances, clinicians may misdiagnose a patient's situation and cause of death. There remains a high discrepancy rate between the ante mortem clinical diagnosis and autopsy diagnosis of cause of death for patients who die in hospitals.[2] Autopsy may be considered valuable in uncovering the most frequent diagnostic pitfalls. There is an increasing need for establishing the exact cause of death which helps in making necessary interventions and proper management of cases.[3] Oral cavity can be described as a window to changes occurring in the human body; almost all systemic variations show manifestations orally. These variations include changes seen in the body due to disease, injury and death.

Also, patients undergoing radiotherapy are subjected to many injurious insults, which manifests orally as erythema, xerostomia radiation caries and osteoradionecrosis. Numerous changes occur in the body after death like cyanosis of mucous membrane, congestion of viscera etc. The changes which occur in the body and oral cavity after death can play a significant key role in determining the cause of death. This particular aspect during postmortem examination is usually overlooked. This article encompasses around a similar concept and reviews the different manifestations in the oral cavity observed post mortem.

Three different subjects with different cause of death

Special reference is given to the significance of oral manifestations and their relation to the cause of death. Post mortem was performed on the dead bodies of three individuals presenting with classical postmortem features of rigor mortis, foul body smell and moderate to intense cyanosis.

Case 1

The first subject was a moderately built, male with approximate age ranging from 30-32 years. Presence of white froth was observed along the corners of the mouth and nose. The eyes displayed a characteristic hazy presentation also referred to as glassy appearance. Examination of oral cavity revealed thickening of the oral mucous membranes with mild whitish discoloration, and moderate to intense white

*Correspondence

Dr. Sneha Sethi

Senior Lecturer,
Sudha Rustagi College of Dental Sciences and
Research, Faridabad, Haryana, India.

E Mail: sneha_sethi123@yahoo.com

discoloration with the attached gingival (fig I). The tongue also displayed white patches. Autopsy findings comprised of congestion with the brain and its meninges, liver, kidney and spleen. Chemical analysis revealed the cause of death to be ingestion of organophosphorus compound. Organophosphorus (OP) compounds constitute a heterogeneous category of chemicals specifically designed for the control of pests, weeds or plant diseases. [11] Organophosphorus compounds produce clinical manifestations by depression of the enzyme cholinesterase resulting in the accumulation of acetylcholine at various receptors. This has three types of effects: (1) muscarinic, (2) nicotinic, and (3) central effects. Muscarinic manifestations include excessive salivation and tracheobronchial secretions (exocrine gland stimulation), miosis and diarrhea (smooth muscle involvement).[5] The sequence of events leading up to death generally consist of inhibition of cholinesterase, acetylcholine accumulation, disruption of nerve function (centrally or peripherally), respiratory failure, and ultimately asphyxiation. It has been estimated that the lethal dose in man is approximately 60g.[6]

The potential adverse impact on human health from exposure to pesticides is likely to be higher in countries like India due to easy availability of highly hazardous products, and low risk awareness, especially among children and women.[7] The WHO estimates, based on 2001 data, that 849,000 people die globally from self-harm each year.[8] The estimated proportion of suicides attributable to pesticide self-poisoning varies considerably across the WHO's six regions: in Europe an estimate of 3.7% of suicides employ pesticides, the Americas: 4.9%, Eastern Mediterranean: 16.5%, Africa: 22.9%, South East Asia: 20.7% and Western Pacific: 55.8%. Organophosphorus insecticides appear to be the most commonly ingested pesticides in rural Asia, accounting for around two thirds of cases.[9] Organophosphorus poisoning; after hanging (49%) is known to be the second most common method of suicide in India (40.3%). The first case of organophosphorus poisoning in India was reported in Kerala in 1958; and it was reported that around 100 people had died.[10]

Case 2

The second subject was a moderately built female, approximately 17-20 years of age. Various patches exhibiting yellowish discoloration were also present all over the body. A 3-4cm wide perforation was seen along the right lower lateral aspect of abdomen. Darkening of the skin was observed from the facial region extending till the neck. Swelling of lips was

presented along with signs of cyanosis. Examination of the oral cavity revealed a faint generalized yellowish tinge with the oral mucosa; the teeth exhibited an unusually white shade and were brittle in consistency (fig II). The autopsy revealed a corroded stomach and perforations in the intestine. The kidney, spleen and liver showed signs of congestion. There was moderate to intense yellowish discoloration of the tongue and mild to moderate discoloration of the esophagus and visceral organs. The chemical analysis in this case showed the cause of death to be sulphuric acid poisoning. The interval of time before death, known as lethal time(LT), after acute ingestion of sulphuric acid is a LT min (minimum time after ingestion of fatal dose of poison that will cause death) of 30 Minutes and an LT50 (time in which 50% of persons will die after ingesting a fatal dose of a Poison) of 5 hours.[11] The mortality due to such causes varies from 4-12%.[12] According to the Holinger and Friedman classification, post-corrosive endoscopic changes are classified on three levels: 1) superficial damage followed by hyperemia, desquamation of the epithelium and edema of the mucous membrane; 2) trans-mucous damage followed by inclusion of all mucous layers, exudation, erosions and ulceration; 3) trans-mural damage followed by penetration of ulceration into the deep-tissue layers and surroundings. The injuries in the mouth, throat, esophagus, stomach and duodenum can be either reversible or irreversible. The damaged mucous membrane, the sub-mucus and muscle layer regenerate only with great difficulty because of the surrounding inflammation, necrosis and secondary complications.[13] Naik SM et al (2012) reported a case of acute accidental formic acid poisoning and examination of the oral cavity revealed intense corrosion of the tongue and the oral mucosa.[14] Vijanath V et al (2010) presented a case of a suicide by consumption of sulphuric acid and the autopsy revealed extensive demarcated cutaneous burns on the inner aspect of the lips.[11] Whereas, Martinez MA et al (2007) reported a case of fatal hydrofluoric poisoning and observed no external burns in the oro-pharynx.[15] In a study performed by Malcol D (1961) on the effect of sulphuric acid on the teeth of battery workers observed the initial lesion to be etching of the labial surface of the enamel, giving a dull ground glass appearance barely visible to the naked eye. The exposed surface of teeth were highly polished or etched. They also said that both material alba and calculus can be dissolved completely from the exposed surfaces in vivo, giving the false presentation of good oral hygiene.[16] Previous studies also showed the increasing brittleness of teeth with exposure to sulphuric acid.[17]Zaheer et al in their study in 2009,

recorded acid ingestion to constitute 25% of all accidental poisonings and no cases of acid poisoning contributed to the homicidal or suicidal category. 8 cases of OPP (organophosphorus poisoning) were observed in the upper middle class, 4 in the lower middle class, 2 in class V or poor and 2 were below poverty line. 2 cases of acid ingestion were recorded in the upper middle class and 2 in class V or the poor category. Acid ingestion tends to occur less frequently in the United States (<5%) but appears to be more common in countries like India where hydrochloric acid and sulfuric acid are easily accessible.[18]

Case 3

The third subject was a well built, well nourished male, with age ranging from 45-50 years. The skin was wrinkled, pale and sodden like a “washer woman’s skin”. The skin of the feet and hands were severely wrinkled and displayed “stocking” and “degloving” presentations respectively i.e. the detachment of the thick keratin of hands and feet which pull off in “glove and stocking fashion. The face was bloated and discolored, along with evidence of fine white froth near the nose and corners of the mouth. Examination the oral cavity revealed congested oral mucosa, and a protruding tongue with fine teeth marks along its lateral margins (fig III). The autopsy revealed congested brain membranes, traces of froth and mud was observed in the larynx and trachea. Lungs were water lodged and voluminous in size, the spleen, kidney, as the cause of death. One of the most classical definitions is provided by Roll: “death by drowning is the result of a hampering of the respiration by obstruction of mouth and nose by a fluid medium (usually water).”[19]Drowning is considered as the

third leading cause of death and the around 388,000 people die due to drowning annually. India along with China together account for 43% of total world’s mortality due to drowning.[19] According to *Lunetta et al* in 2002, on external examination, the plume of froth at the mouth and nostrils can be considered as a valuable indication. The pitfall is that this finding is non-specific, quite transient and can only be found in fresh drowned bodies.[20] *Farrugia A* and *Ludes B* also stated that one of the signs of drowning would be large amounts of froth present around nostrils and mouth in freshly drowned bodies.[21] *Carter N et al* in 1998 suggested that the interpretation of hemorrhages in the neck tissues can be controversial. In the absence of trauma in that region, those bleedings may most probably be due to hypostasis or to an extension or the Prinsloo and Gordon artifact (postmortem hemorrhage on the posterior surface of the esophagus). In a minority of the cases, these hemorrhages can be explained by violent neck movements during the drowning process.[22] *Bohnert et al* studied the liquid amounts in the sphenoid sinuses: the average volume of the aspirate was smaller in the control group than in the drowning victims.[23] *Hotmar* found that both the investigation of liquid in sphenoid and maxillary sinuses can provide auxiliary information in the diagnosis of drowning.[24] *Papadodima et al* in their research observed that the strongest pathognomonic finding for drowning on autopsy was frothy fluid in the oral cavity and respiratory tracts. Unfortunately, it was rarely seen as it is expelled by attempts at cardiopulmonary resuscitation or when the body is moved for transportation to the morgue.[25]



Fig I: Oral manifestations in the case of organophosphorus poisoning

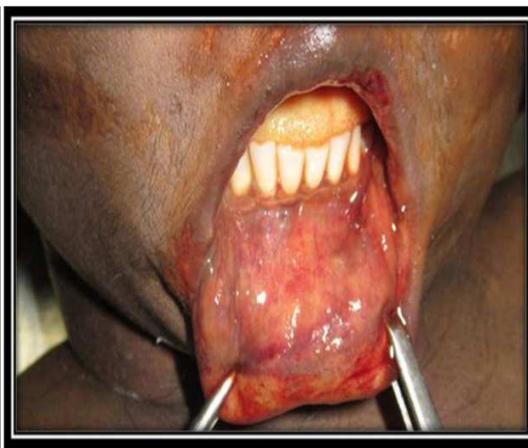


Fig II: Oral manifestations in the case of sulphuric acid poisoning



Fig III: Oral manifestations in the case of drowning

Review

A general review of previous literature showed us many cases with documented post-mortem oral manifestations. *Stewart I et al* [26] reported findings of recreational and occupational field exposure to freshwater cyanobacteria, and in his review they documented some common manifestations such as pneumonia and blistering of the mouth. *Papadodima SA et al* [25] in their original research on cardiovascular disease and drowning, stated that the strongest pathognomonic findings for drowning on autopsy is frothy fluid in the oral cavity and respiratory tracts. *Gallagher KE et al* [27] reported a case of suicidal asphyxiation in which the nares contained frothy white edema fluid, and gray-white frothy edema fluid was also found to be protruding from the oral cavity. *Gerhardsson L et al* [28] presented a case report on a case of fatal arsenic poisoning and on postmortem examination, moderate edema was seen in the mucosa of the piriform recess on both sides, while the epiglottis was normal. *Benecke M* [29] presented a review on the history on forensic entomology, and found that German doctors had conducted previous forensic insect studies and one of those studies presented a report of a child succumbing to insect bites. The autopsy of the child revealed patches on the nose and lips, proceeding downwards from the child's mouth. Also the tongue was not discolored but did show bleeding of the tip. *Introma F et al* [30] presented three cases with regard to forensic entomology and in one of the cases; the murder victim had been burnt to death to conceal the true cause of death, examination of the remains during autopsy revealed two bullet entrance wounds in the neck. The bullets were shown by postmortem

radiographs of the skull; first within the oral cavity close to the left angle of the mandible and the second the cranial cavity. The cause of death was recognized by dental record comparison. *Silva RF et al* [31] reviewed different cases in which smile photograph analyses were found to be crucial in determining the positive identification of the unidentified human bodies. All of the cases were subjected to personal identification by photographs of smile including one adult male found in an advanced stage of decomposition, one adult female disappearing during an ecotourism trip, and one carbonized body of a male individual found in a forest region. During the autopsy the photographs of the smiles were used by comparison of the ante and postmortem images giving accurate and useful information not only about dental state but also about the anatomical features surrounding the upper and lower anterior dental arches. They concluded that this method was not time-consuming and also had the advantage of allowing extra oral dental examination. *Munnynck KD et al* [32] presented a review on the forensic approach of fatal dog attacks, and they observed that in the group of children younger than 4 years, 63% of bites were to the face, head and neck, usually severe and often even fatal. They stated that most fatal lesions are located in the head and neck region involving soft tissue laceration, larynx crushing as well as opening or compression of the extra cranial vessels, causing death by means of asphyxia or hemorrhage. They also reported a case of a fatal dog attack on a 6 year old girl. On postmortem external examination they observed extensive abrasions on the face with bruised lips and petechial hemorrhages on

the conjunctival mucosa of the lower eyelids. *Hitosugi M et al* [33] reported a case of fatal benzalkonium chloride poisoning, the autopsy revealed corrosive changes of the mucosal surfaces of the tongue, pharynx, larynx, esophagus and stomach. The lips and oral cavity were intensely erythematous and could be easily removed. *Sweet D et al* [34] reported case where physical evidence in the form of high quality bite mark was discovered on a piece of yellow cheese found at the crime scene. *Pumphrey RSH et al* [35] reviewed the postmortem findings after fatal anaphylactic reactions and observed cutaneous erythema/urticaria, laryngeal or pharyngeal edema. They also noted that upper airway edema was more common in deaths related to food allergens than in those after reactions to venom or drugs. Chelioscopy and Palatoscopy are also established as significant in identification especially in burn victims, lip grooves and palatal rugae are used successfully in human identification.[36-37] The success rate of dental identification varies considerably depending on the nature of the accident, the nationality and country of residence of the victims, the incidence of dental treatment, the availability of adequate dental records, and the degree of dental injuries.[38]

Conclusion

The cause of death may be achieved after considering all the forensic investigations i.e., external examination, autopsy findings, histological and toxicological analysis, blood analysis, biochemical analysis etc. The oral cavity can be considered as a region with tremendous untapped potential especially in regard to coming to a final forensic diagnosis, but this aspect of forensic medicine is relatively undiscovered and overlooked. The above cases shed light on the prospects offered by observation of the manifestations of the oral cavity post mortem and their significance in the field of forensic science.

References

1. Nandy A. Principles of forensic medicine. 3rd ed. Calcutta: New central book agency (p) ltd; 2005.
2. Spiliopoulou C, Papadodima S, Kotakidis N et al. Clinical diagnosis and autopsy findings: a retrospective analysis of 252 cases in Greece. Arch Pathol Lab Med 2005 Feb;129:210-14.
3. Aguirre OJM, Camacho VG, Lopez LG, Gonzalez GA, Nava GJI. Concordance between premortem and postmortem diagnosis in the autopsy : result of a 10 year study in a tertiary care centre. Ann Diagn Pathol 2003; 7(4):223-30.
4. Kumar SV, Fareedullah M et al. Current review on organophosphorus poisoning. Arch Appl Sci Res 2010;2(4):199-215.
5. Wadia RS, Sadagopan C, Amin RB and Sardesai HV. Neurological manifestations of organophosphorus insecticide poisoning. J Neurol Neurosurg Psychiatry 1974 July;37(7):841-847.
6. Jadhav RK, Sharma VK, Rao GJ, Saraf AK and Chandra H. Distribution of malathion in body tissues and fluids, Forensic Sci Int 1992;52:223–229.
7. Eddleston M. Buckley NA, Eyer P and Dawson AH. Management of acute organophosphorus pesticides poisoning. Lancet. 2008 Feb 16; 371(9612):597–607.
8. Mutangadura, Gladys B. The World Health report 2002. Reducing risks, promoting healthy life. Blackwell 2004
9. Gunnel D, Eddleston M, Phillips MR and Konradsen F. The global distribution of fatal pesticides self-poisoning: Systematic review. BMC Public Health 2007;7(357):1-15.
10. Aktar WA, Sengupta D and Chowdhury A. Impact of pesticides use in agriculture: their benefits and hazards. Interdiscip Toxicol 2009 March;2(1):1–12
11. Vijayanath V, Raju GM, Rao KN and Anitha MR. Suicidal Acid injury; a case report. J Indian Acad Forensic Med 2010;32(4):347-348.
12. Vij K. Textbook of forensic medicine and toxicology: principles and practice. 4th ed. Elsevier 2008.
13. Cibisev A, Nikolova-Todorova Z, Bozinovska C, Petrovski D and Spasovski G. Epidemiology of severe poisonings caused by ingestion of caustic substances. Contributions Sec Biol Med Sci 2007;28(2):171–183.
14. Naik SM, Ravishankara S, Appaji MK, Goutham MK, Devi NP, Mushannavar AP et al. Acute accidental formic acid poisoning: a common problem reported in rubber plantations in Sulia. Int J Head Neck Surg 2012;3(2):101-105.
15. Martínez MA, Ballesteros S, Piga FJ, Sánchez de la Torre C, Cubero CA. The tissue distribution of fluoride in a fatal case of self-poisoning. J Anal Toxicol. 2007 Oct;31(8):526-33.
16. Malcol D and Paul E. Erosion of the teeth due to sulphuric acid in the battery industry. Brit J Industr Med 1961;18(6):63-69.
17. Misch J (1923). Lehrbuch der Grenzgebiete der Medizin und Zahnheilkunde, 3rd ed. Vogel, Leipzig.
18. Zaheer MS, Aslam A, Gupta V, Sharma V and Khan SA. Profile of poisoning cases at a north

- Indian tertiary care hospital. Health and Population, perspectives and issues 2009;32(4):176-183.
19. Piette MHA and Letter EAD. Drowning : still a difficult diagnosis. *Sci Int* 2006;163:1-9.
 20. Lunetta P, Penttila A, Sajantila A, Circumstances and macropathologic findings in 1590 consecutive cases of bodies found in water. *Am J Forensic Med Pathol* 2002;23:371–376.
 21. Audrey Farrugia and Bertrand Ludes. Diagnostic of Drowning in Forensic Medicine, Forensic Medicine – from Old problems to New Challenges, Prof. Duarte Nuno Vieira (Ed.)2011.InTech.
 22. Carter N, Ali F, MA. Green, Problems in the interpretation of hemorrhage into neck musculature in cases of drowning, *Am J Forensic Med Pathol* 19 (1998) 223–225.
 23. Bohnert M, Ropohl D, Pollak S. Zur rechtsmedizinischen Bedeutung des Flußsigkeitsgehaltes in den Keilbeinhöhlen, *Arch. Kriminol* 209(2002)158–164.
 24. Hotmar P. Nachweis von Flußsigkeit in den Nasennebenhöhlen als mögliches diagnostisches Zeichen des Ertrinkungstodes, *Arch. Kriminol.* 198 (1996) 89–94.
 25. Papadodima SA, Sakelliadis EI, Kotretsos PS, Athanaselis SA and Spiliopoulou CA Cardiovascular disease and drowning: autopsy and laboratory findings. *Hellenic J Cardiol* 2007 Jul-Aug;48(4):198-205.
 26. Stewart I, Webb PM, Schluter PJ and Shaw GR. Recreational and occupational field exposure to freshwater cyanobacteria – a review of anecdotal and case reports, epidemiological studies and the challenges for epidemiological assessment. *Environmental Health: A Global Access Science Source* 2006;6:1-13.
 27. Gallagher KE, Smith DM, Mellen PF. Suicidal asphyxiation by using pure pure helium gas - Case report, Review and discussion of the influence of the internet. *Am J Forensic Med Pathol* 2003;24:361-363.
 28. Gerhardsson L, Dahlgren E, Eriksson A, Lagerkvist BEA, Lundstrom J and Nordberg GF. Fatal arsenic poisoning – a case report. *Scand J Work Environ Health* 1988;14(2):130-133.
 29. Benecke M. A brief history of forensic entomology. *Forensic Sci Int* 2001;120:2-14.
 30. Introma F, Campobasso and Fazio AD. Three case studies in forensic entomology from southern Italy. *J Forensic Sci* 1998;43(1):210-214.
 31. Silva RF, Pereira SD, Prado FB and Daruge E. Forensic odontology identification using smile photograph analysis – case reports. *J Forensic Odontostomatol* 2008;27:1:12-17.
 32. Munnynck KD and Voorde WV. Forensic approach of fatal dog attacks : a case report and literature review. *Int J Legal Med* 2002;116:295-300.
 33. Hitosugi M, Maruyama K and Takatsu A. A case of fatal benzalkonium chloride poisoning. *Int J Legal Med* 1998;111:265-266.
 34. Sweet D and Hildebrand D. Saliva from cheese bite yields DNA profile of burglar : a case report. *Int J Legal Med* 1999;112:210-203.
 35. Pumphrey RSH and Roberts ISD. Postmortem findings after fatal anaphylactic reactions. *J Clin Pathol* 2000;53:273-276.
 36. Caldes IM, M aghalhaes T and Afonso A. Establishing identity using chelioscopy and palatoscopy. *Forensic Sci Int* 2007;165(1):1-9.
 37. Muthusubramanian M, Limson KS and Julian R. Analysis of rugae in burn victims and cadavers to simulate rugae identification in cases of incineration and decomposition. *J Forensic Odontostomatol* 2005;23:26-29.
 38. Aurora V, Tomas M, Ncomedes E, Heras MDL, Stella and Gonzalo G. Comparative study of efficiency of dental methods for identification of burn victims in two bus accidents in Spain. *Am J Forensic Med Pathol* 2002;23(4):390-393.

Source of Support: Nil

Conflict of Interest: None