

Original Article

DIAGNOSTIC EFFICACY OF CARDIAC ISOENZYME CK-MB IN PERICARDIAL FLUID FOR POSTMORTEM DIAGNOSIS OF MYOCARDIAL INFARCTION

Dr. PS Ghormade, Dr. NB Kumar, Dr. CV Tingne, Dr. AN Keoliya

Authors

Dr.Pankaj Suresh Ghormade, Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr.NarendraBaluram Kumar, Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr.ChaitanyaVidyadharTingne, Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr.Ajay Narmadaprasad Keoliya, Professor and Head, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Number of pages: Eight

Number of Tables: Five

Number of Graph: One

Corresponding Author: Dr. Pankaj Suresh Ghormade
At.668 B ,OppositeHansveni Guest House ,
Karnewarmarg ,Telipura ,Sitabuldi ,Nagpur
Maharashtra, India-440012
pankajforensic@gmail.com, 918888839497

Original Article

DIAGNOSTIC EFFICACY OF CARDIAC ISOENZYME CK-MB IN PERICARDIAL FLUID FOR POSTMORTEM DIAGNOSIS OF MYOCARDIAL INFARCTION

Dr. PS Ghormade, Dr. NB Kumar, Dr. CV Tingne, Dr. AN Keoliya

Abstract

Sudden cardiac death due to acute myocardial infarction is the most prevalent cause of death in adults and constitutes a significant portion of the autopsies that are conducted by Forensic pathologists in our country. Serum cardiac isoenzymes creatine phosphokinase-MB (CK-MB) has high sensitivity and specificity for cardiac damage and it is routinely used for clinical diagnosis of myocardial infarction (MI). However, in forensic medicine, diagnostic utility of this cardiac marker for postmortem diagnosis of MI has not been fully established. Hence, present study is carried out with aim to evaluate the diagnostic efficacy of CK-MB in pericardial fluid for autopsy diagnosis of MI. The present study included 119 medico-legal autopsy cases selected during a period of 2 years. Four study groups were formed depending upon final cause of death as follows: (1) Sudden cardiac death due to Ischemic heart disease (IHD) (n= 52), (2) violent asphyxia (n=24); (3) Polytraumatic deaths (n=20); (4) natural deaths excluding cardiac causes (n=23). Biochemical analysis of pericardial fluid was carried out. Highest levels of CK-MB were noted in deaths due to IHD. By using this cardiac marker, early infarcts can be predicted in sudden cardiac deaths associated with severe coronary artery disease & inconclusive histopathological findings. We obtained high diagnostic sensitivity and negative predictive value of CK-MB for autopsy diagnosis of MI in pericardial fluid comparable to the clinical analyses on serum.

Key words: Autopsy, IHD, Forensic pathology, myocardial infarction, pericardial fluid, CK-MB.

Introduction

Ischemic heart disease (IHD) due to coronary atherosclerosis is the most prevalent cause of sudden death in adults over the age of 30 years, but it is not infrequent in younger subjects.¹ Hence, Coronary artery disease is sometimes called 'The Captain of the Men of death' and it constitute a significant portion of the autopsies that are conducted by Forensic Pathologists in our country.² 'Sudden cardiac death' is defined as, natural death due to cardiac causes, heralded by abrupt loss of consciousness within 1 h of the onset of acute symptoms.³ In more than 50% of these cases, death is caused due to cardiac arrhythmias induced by myocardial ischemia. And when autopsy is performed, there may be presence of coronary atherosclerosis without evidence of demonstrable gross or microscopic findings of myocardial infarction (MI). As survival period of more than 6 hours is required for changes of MI to appear in the heart, infarction is not apparent on gross examination until 12–24 hrs and light microscopic (H&E) changes are not apparent before 4–6 hrs.⁴ Due to limitations of histopathological findings⁵, it is necessary to establish diagnostic utility of different biochemical cardiac markers in biological fluids for postmortem diagnosis of MI. Measurements of CK-MB and other cardiac markers in serum are commonly used for clinical diagnosis of acute myocardial infarction⁶⁻⁸. Various authors have investigated role of CK-MB for postmortem diagnosis of MI in serum and pericardial fluid (PF).⁹⁻¹⁸ Few of them have attempted to determine if postmortem levels of this isoenzyme is significantly higher in deaths because of myocardial ischemia than those because of other causes of death.⁹⁻¹³ However, these studies do not mention the overall diagnostic efficacy of this marker as

mentioned in clinical practice. Hence, in the present study, we had investigated diagnostic efficacy & distribution of CK-MB in PF fluids for autopsy diagnosis of MI as compared to other causes of death.

Material & Methods

In this prospective study, total 119 cases (91 males and 28 females) were studied from total medico legal autopsies from Dec-2010 to June-2012 conducted at dept. of Forensic Medicine, IGGMC, Nagpur. Mean age of subject was 50.77 years (S.D.10.81, range 30-75 years). Mean post-mortem interval was 13.50 hours (S.D.6.90 hours, range 2-24 hours). All dead bodies were kept in cold compartments prior to autopsy. We have excluded from the study all cases with pericardial disease or haemorrhage in pericardial fluid & cases showing signs of decomposition.

Subjects were assigned into one of the four diagnostic groups depending upon their final cause of death as follows:

Group I - Sudden cardiac deaths due to Ischemic Heart Disease (n=52) subdivided in deaths due to myocardial infarction (n=28) and coronary artery disease (CAD) (n=24). These subgroups were classified upon histopathological confirmation.

Group II – Violent Asphyxial Deaths (n=24) subdivided into deaths due to hanging (n=16) and drowning (n=8).

Group III – Deaths due to Polytrauma (n=20) included vehicular accident cases with extensive muscle damage without any evidence of chest trauma.

Group IV - Natural deaths not due to cardiac diseases (n=23) included deaths due to pulmonary consolidation (n=10), lung abscess (n=04), non-traumatic intracerebral hemorrhage (ICH) (n=04), non-traumatic subarachnoid hemorrhage (SAH) (n=02), sepsis due to infected wounds (n=02) and acute bronchial asthma (01).

Details in each case were obtained from inquest paper, treatment record, death certificate and other relevant documents issued from hospitals in hospitalized cases. In addition, history particularly important towards diagnosis of cardiac disease was elicited from near relatives or friends. For studying factors affecting CK-MB levels in pericardial fluid, we have included cases of polytrauma to analyse effect of muscle damage and cases of violent asphyxia to observe the effect of hypoxia on heart and consequent release of cardiac marker. Whereas, other natural deaths in group IV were assigned as controls.

Survival time (ST) was known in 78 cases & mean ST was 1.21 hours (S.D. 1.01 hours, range=0.10-5 hours). All cases in group II were brought dead to the emergency department. Out of total 119 cases, Cardio pulmonary resuscitation (CPR) was attempted in 45 cases. In group I, cardiac complaints were present in 43 cases, 21 patients were hospitalized & ECG findings of IHD's were present in 8 of these cases.

Pericardial fluid samples were collected from pericardial sac by using sterile syringe after incising parietal pericardium. It was then centrifuged immediately at 5000 rpm for 15 minutes & supernatant was collected for enzyme analysis. Standard laboratory procedures were used for measuring levels of CK-MB in pericardial fluid & biochemical analysis was carried out on an automated analyser using commercial kits. We used Immunoinhibition/Mod.IFCC⁸ method of estimation for CK-MB involving principle of UV kinetic reaction. As the PF showed high enzymatic activities compared to clinical range on initial tests, dilution of fluid was carried with normal saline (0.9%) in the ratio of 1:9 before analysis and results then obtained were multiplied by 10.

Detailed gross examination and dissection of the heart was carried out by short-axis and inflow outflow techniques. Only those cases showing severe coronary artery disease i.e. vessel showing $\geq 75\%$ stenosis of lumen on transverse sectioning¹⁹, were included in final

observations. Histopathological examination (HPE) of heart was carried out in each case with haematoxylin and eosin (H&E) staining. On HPE, findings of MI were divided into acute, healing and healed infarct. Hearts showing presence of coagulative necrosis with various degrees of nuclear changes and prominent infiltration of neutrophils were diagnosed as acute MI (AMI). Whereas, cases in which heart showed, above changes in various stages with presence of mononuclear leucocytes and fibroblasts without neutrophils were considered as healing MI (HMI). When, HPE of hearts showed presence of collagenous scarring without cellular infiltration, cases were labeled as old healed MI (OHMI). While, presence of features like interfibrillar edema/interstitial edema and patchy eosinophila were considered as inconclusive and cases were assigned into deaths due CAD.

Statistical analysis

For statistical analyses of the data, the MedCalc version 13.1.0.0 program was used. Probability level $p < 0.05$ was considered significant. Non-parametric tests i.e. Kruskal–Wallis test & Mann–Withney test (rank–sum test) were used to compare levels of CK-MB in PF amongst four diagnostic groups & to compare pair of diagnostic groups, respectively. In addition, specific contrasts for variable grouped by diagnostic category were carried out using Mann–Withney test. Receiver operating characteristic (ROC) curve^{20,21} was used for measuring area under the curve for CK-MB, to obtain its cut-off level for evaluating diagnostic efficacy and to discriminate between cases died due of IHD’s and non -IHD by using diagnostic cut off value.

The study protocol was approved by the Institutional Ethical Committee.

Observations & Results:

All 52 cases included in group I, had severe coronary artery atherosclerosis in one or more major epicardial arteries and triple vessel disease pattern was predominantly (48.07%) seen. Coronary thrombosis was found in 7 cases of acute MI. Table.1 shows histopathological findings in heart from all cases in diagnostic groups.

Table 1: Histopathological findings of heart in diagnostic groups

Groups	Total cases	AMI or HMI	AMI+ OHMI	OH MI	MF	Interstitial edema	Congestion
I (IHD)	52	AMI-19 HMI-4	5	14	2	46	52
II (Violent asphyxial deaths)	24	-	-	2	0	10	24
III (Polytraumatic deaths)	20	-	-	-	0	0	20
IV (Non-cardiac natural deaths)	23	-	-		3 (2 ICH, 1 SAH)	3	23

HMI-Healing MI, OHMI-Old Healed MI, MF-Myocardial Fibrosis

Table 2.shows the values (Mean±Standard error of mean, S.D. and range) obtained for CK–MB in each diagnostic group. Highest levels were observed in death due to IHD’s as

compared to cases in other diagnostic groups. Non-parametric Kruskal–Wallis test (Table 2) was used for comparing differences in CK-MB level in PF amongst all diagnostic groups. We have observed statistically high significant difference in activities of CPK-MB (P=0.0001) amongst all four diagnostic groups.

Table 2: Pericardial fluid levels of CK-MB and results of Kruskal-Wallis test in the diagnostic groups

Parameter	Levels	I. Ischaemic heart Disease	II. Violent asphyxia	III. Polytrauma	IV. Non cardiac natural deaths	Result of Kruskal-Wallis test
CK-MB (U/L)	Mean ±SEM	4635.36 ± 713.	1623.16 ± 442.3	1088.2 ± 280.9	858.67 ±313.5	df=3 test statistics (t)=48.50 P-value=0.0001
	S.D.	5144.92	2166.99	1256.12	1503.65	
	Median	2176.9	458.75	396.5	310	
	Range	497.82- 18943.	78 – 8480	52- 4780	115 -6280	

SEM: Standard Error of Mean, df: degree of freedom

In group I, statistically non-significant (P=0.6729) differences were observed in values (Table.3) of CK-MB between cases of definite MI (n=28) & cases with CAD (n=24) .On the Mann-Whitney test (Table no.4), we observed highly significant (P<0.0001) levels of CK-MB in cases of death due IHD's as compared to deaths due to violent asphyxia, polytrauma and other natural deaths excluding cardiac causes.

Table 3: The Mann–Withney test showing difference in levels of enzymes in cases of definite MI & cases of CAD in group-I

Group I (IHD's)	CK-MB (U/L)	
	Myocardial infarction (n=28)	Mean
SD		5968.75
Median		2113.1
Range		497.82 – 18943.6
Coronary artery disease (n=24)	Mean	4144.77
	SD	4050.5
	Median	2405.5
	Range	1018 – 15618
	Z statistics	0.422
	P value	0.6729

Table 4: Mann–Withney test between diagnostic groups

Variable	Comparison groups	Z statistic Value	P value
CK-MB	I – II	4.101	0.000
	I – II	4.564	0.000
	I – IV	5.538	0.000

(Groups: I–IHD, II-Violent asphyxia, III-Polytrauma, IV-Natural deaths other than cardiac disease)

Receiver-operating characteristic (ROC) curve analysis:

For discriminant analyses, we used cause of death as grouping variable, total 119 cases in all groups were divided into deaths due to IHD's (n= 52) and that of due to non IHD's (n=67).ROC curve (**Fig.1**) was established by taking levels of CK-MB as an

independent variable with paying special attention to the area (Table 5), which represents the correct diagnosis in two individuals, one with MI and one without MI. By using ROC curve, we determined the diagnostic cut-off value of 979 U/L for CK-MB in pericardial fluid (Table 5) for post-mortem diagnosis of myocardial infarction in cases of IHD's.

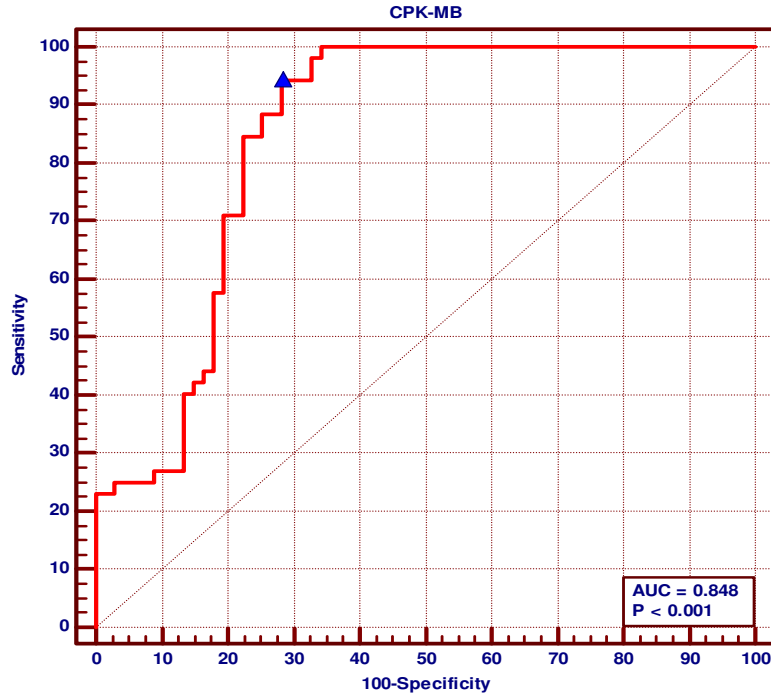


Fig.1: ROC curve of CK-MB levels in pericardial fluid

At this diagnostic cut off level, we have obtained sensitivity=94.23% NPV=94.1% & specificity=71.64% of CK-MB for diagnosis of MI. No statistically significant correlations were observed between the levels of CK-MB and postmortem interval period included in the study and the use of cardiopulmonary resuscitation.

Table 5: Area under curve & diagnostic cut-off levels of CK-MB established using ROC curve analysis

Variable	AUC*	P	SE**	Lower limit	Upper limit	Cut-off level	Sensitivity	Specificity	NPV#
CK-MB	0.848	0.001	0.036	0.771	0.907	979 U/L	94.23%	71.64%	94.1%

*Area under curve, ** Standard error, #Negative predictive value

Discussion

As mentioned in literature, following acute myocardial infarction, the initial CK-MB rise occurs 4 to 6 hours after the onset of chest pain, peaks at 24 hours, and returns to baseline at 48 to 72 hours (t1/2 of CK-2 is 10 to 12 hours).One advantage of CK-MB over other markers is that it remain elevated for longer periods and it is easier to detect reinfarction using serial measurement.⁶⁻⁸ In clinical practice, measurement of CK-MB level in serum is routinely used to detect myocardial ischemia. However, use of this cardiac marker in serum

for postmortem diagnosis of MI has limitations due other factors affecting enzyme levels.^{11,13} Hence, we chose pericardial fluid over other biological fluids because, it is an ultrafiltrate of plasma^{22,23} hence, biochemical analysis can be done by using kits standardized for serum. It lacks RBC's, therefore does not show haemolysis phenomenon that frequently interfere with biochemical determinations in serum. And as pericardial and myocardial irrigations are shared, markers of myocardial ischemia are detectable in PF before they are detectable other biological fluids.⁹⁻¹⁸

We obtained highest values of CK-MB in group of deaths due to IHD's as compared to other diagnostic groups. Statistically non-significant differences were noted in levels of CK-MB between cases of AMI and cases with inconclusive H&E findings classified as deaths due to severe coronary atherosclerosis in group I. This signifies utility of performing CK-MB test on pericardial fluid for post-mortem diagnosis of early MI, because histopathological finding may be inconclusive in such cases. This finding from our study concur with the findings of earlier studies.^{13,25}

On Kruskal-Wallis test, highly significant differences were observed for CK-MB amongst all diagnostic groups. On Mann-Whitney test, we observed statistically significant levels of CK-MB in group of subjects who died of IHD's (Group-I) in comparison to other groups represented by the subjects who died due to violent asphyxia (Group-II), polytrauma (Group-III) and natural deaths excluding cardiac causes (Group-IV). Findings of our study are in accordance with ones that reported in earlier studies.^{9,10,15,17,25} However, Barabas B¹⁸ found non-significant difference in CK-MB levels between asphyxial deaths and death due to AMI citing intense agony prior to death as probable reason for conflicting results. On ROC curve analysis, we have observed significant areas under the curve for CK-MB (0.848). Diagnostic cut-off point value obtained for CK-MB showed very high sensitivity and negative predictive value and only 3 cases out of 52 included in IHD's, had values less than cut-off obtained. These are the cases of MI in healing phase, suggestive of probable decline in enzyme levels after its initial peak during acute phase. Whereas, 19 cases of non-IHD out of 67 were incorrectly classified leading to lower specificity for the marker. This might be due to other factors affecting its levels, as out of those 19 cases, 9 corresponded to violent asphyxia deaths in which there might well have been an intense agony with consequent acute myocardial suffering involving the release of different markers into the cadaver, another 7 had died from polytraumatism in which, too, cardiac traumatism may have been involved. 3 deaths were from natural causes (2-ICH and 1-SAH) that showed presence of severe coronary artery disease and death may occur due to cardiac arrhythmias in these disease conditions.^{26, 27} We obtained higher diagnostic sensitivity and NPV for CK-MB on ROC curve analysis as compared with the results of Carceles-Perez et al¹⁰. In clinical practice, sensitivity of the serum CK-MB for diagnosis of MI is from 92 to 100 at 3 hours from the onset of symptoms, whereas diagnostic specificity has been reported to be very close to 100%.²⁸

Conclusion

As we obtained nearly equal diagnostic sensitivity and negative predictive value for the cardiac marker CK-MB in pericardial fluid compared to clinical analysis on serum sample, hence, this cardiac marker in pericardial fluid could be of great help for autopsy diagnosis of MI in combination with histopathological examination.

References

1. Thiene G, Basso C, Corrado D. Cardiovascular causes of sudden death. In: Silver MD, Gotlieb AI, Schoen FJ, eds. Cardiovascular Pathology. Philadelphia: Churchill Livingstone; 2001;p-326.
2. De la Grandmaison GL. Is there progress in the autopsy diagnosis of sudden unexpected death in adults? *Forensic Sci Int.* 2006; 156:138–44.
3. Gupta R, Joshi P, Mohan V, et al. Epidemiology and causation of coronary heart disease and stroke in India. *Heart.*2008; 94:16-26.
4. Baroldi G. Myocardial cell death, including ischemic heart disease and its complication. In: Silver MD, Gotlieb AI e Schoen FJ, eds. Cardiovascular Pathology. Philadelphia: Churchill Livingstone; 2001:198
5. Saukko P, Knight B. The pathology of sudden death. In Saukko P, Knight B eds. *Knight's Forensic Pathology.*3rd ed. Hodder Arnold.2004; pp- 492-501.
6. Adams JE, Abendschein DR, Jaffe AS. Biochemical markers of myocardial injury: is MB creatine kinase the choice for the 1990s? *Circulation.*1993;88:750-63.
7. Lewandrowski K, Chen A, Jannuzzi J. Cardiac markers for myocardial infarction – a brief review. *Am J Clin Pathol.* 2002;118 (1):S93-S99
8. Henderson AR, Moss DW. Enzymes.In: Burtis CA, Ashwood ER, eds. *Tietz Textbook of Clinical Chemistry.* 5th edn. Philadelphia, Saunders Co., 1986; pp.356-366
9. Osuna E, Carceles-Perez DM, Vieira DN, Luna A. Distribution of biochemical markers in biologic fluids: application to the postmortem diagnosis of myocardial infarction. *Am J Forensic Med Pathol.* 1998; 19(2):123–8.
10. Carceles-Perez DM, Noeguera J, Jimenez JL, Martinez P, Luna A, Osuna E. Diagnostic efficacy of biochemical markers in diagnosis post-mortem of ischemic heart disease. *Forensic Sci Int.* 2004; 142(1):1–7.
11. Luna A, Villanueva E, Castellano MA, Jimenez G. The determination of CK, LDH and its isoenzymes in pericardial fluid and its application to the post-mortem diagnosis of myocardial infarction. *Forensic Sci Int.* 1982; 19:85-91.
12. Luna A, Carmona A, Villanueva E. The postmortem determination of CK isoenzymes in pericardial fluid in various causes of death. *Forensic Sci Int.* 1983; 22:23-30
13. Stewart RV, Zumwalt RE, Hirsch SC, Kaplan L. Postmortem diagnosis of myocardial disease by enzyme analysis of pericardial fluid. *Am J Clin Pathol.*1984; 82:411–17.
14. Osuna E, Carceles-Perez DM, Jakobsson SW, Luna A. Biochemical and morphological markers in the postmortem diagnosis of ischemic heart distress. *Acta Med Leg Soc (Liege).*1990; 40: 275- 83.
15. Burns J, Milroy CM, Hulciewicz B, West CR, Walkley SM, Roberts NB. Necropsy study of association between sudden death and cardiac enzymes. *J ClinPathol.*1992; 45:217- 20.
16. Carceles-Perez DM, Osuna E, Viera ND, Martinez A, Luna A. Biochemical assessment of acute myocardial ischaemia. *J Clin Pathol.*1995; 48: 124-28.
17. Batalis IN, Marcus JB, Papadea NC, Collins AK .The role of postmortem cardiac markers in diagnosis of acute myocardial infarction. *J Forensic Sci.* 2010; 55(4):1088-91
18. Barabas B. CK-MB study in myocardial infarction in sudden cardiac death. *Rom J Leg Med.*2009;17 (2):156-62
19. Basso C, Burke M, Fornes P et al. Guidelines for autopsy investigation of sudden cardiac death. *Virchows Arch.*2008; 452: 11-8.
20. Zweig HM, Campbell G. Receiver-operating characteristic (ROC) plots: fundamental evaluation tool in clinical medicine. *Clin Chem.*1993;39: 561–77.
21. Fan J, Upadhye S, Worster A. Understanding receiver operating characteristic (ROC) curves. *Can J Emerg Med.*2006;8(1):19-20

22. Ben-Horin S, Shinfeld A, Kachel E, Chetrit A, Livnysieh A. The composition of normal pericardial fluid and its implications for diagnosing pericardial effusions. *Am J Med.* 2005; 118:636–40.
23. Gibson TA , Segal BM. A study of the composition of pericardial fluid with special reference to the probable fluid formation. *J Physiol.* 1978,277: 367-77.
24. Sanchez-Martinez CM, Vicente-Rodriguez C. Sudden death: correlation histopathological and biochemical. *Forensic Sci Int.* 2004; 146:S31-32
25. Hopster JD, Milroy MC, Burns J, Roberts BN. Necropsy study of the association between sudden cardiac death, cardiac isoenzymes and contraction band necrosis. *J Clin Pathol.*1996; 49: 403-06
26. Smith M, Rag CT. Cardiac arrhythmias, increased intracranial pressure and the autonomic nervous system. *Chest.*1972, 61:125-33.
27. Vidal BE, Dergal EB, Cesarmen E, et al. Cardiac arrhythmias with subarachnoid haemorrhage. *Neurosurgery.*1979,5:675-80.
28. Kemp M, Donovan J, Higham H, Hooper J. Biochemical markers of myocardial injury. *British J of Anaes.*2004; 93 (1): 63-73.

5. As this study is done in Central India region the application of standards of this study may be considered ideal for application in the region of Central India.
6. Population in Central India is mixed type comprising of various religions and castes, so this study is not applicable to specific caste or religion for estimation of age.
7. Due to changing life style pattern, dietary, climatic, behavioral factors; age of ossification is changing as mentioned in the available literature. So as to evaluate these changes, studies are recommended in every region of India at regular time period for academic and judicial interest.
8. Due to very narrow borderline range of differentiation between various stages of fusion, it is difficult to consider stage of fusion as age indicator.
9. For Radiological study, proper exposure of X-ray, proper positioning while X-ray shooting and proper development of digital X-rays (DX) is necessary.
10. Radiological interpretations are observer dependent so the set standards should be considered under expert guidance to arrive at conclusion in such radiological studies.
11. Along with clinical and dental examination, radiological study plays an important role to arrive at the opinion about the age in medicolegal cases.
12. The opinion about age should always be given in the range. From this study, range of 1-2 years of margin of error can be concluded.
13. For estimation of age relevant joints should be radiologically examined for different centres and opinion should be arrived considering the status of multiple centers.
14. With similar findings we have observed, there is enhancement of belief in the theory that the similarities in geographic-climatic condition, ethnicity, socioeconomic status, dietary habits have the common influence on the fusion of epiphysis with the age.

Limitations of study:

1. Population in Central India is mixed type comprising of various religions and castes so this study is not applicable to specific caste or religion.
2. Dietary, religious, economic, environmental factors are not studied in the present context.
3. As the number of subjects were less, for confirmation of various variations, more studies are required.

References:

1. Aggarwal A. Ages of ossification-Personal Identification in Self-Assessment and Review of Forensic Medicine and Toxicology. 1sted. Delhi: Jaypee Publishers and Distributers (P) Ltd.; 2006. P.51-59.
2. Alcazar ML, Alvear J, Muzzo S. Influence of Nutrition on the bone-development in the child. Archivos Latinoamericanos De Nutrition 1984; 34 (2): 298-307.
3. Bajaj ID, Bharadwaj OP, Bharadwaj S. Appearance and fusion of important ossification centers - A study in Delhi population. Indian J. Med Res. 1967; 55:1064 – 1067.
4. Bargoitra RN, Bargoitra M and Singh J. The time of fusion of medial epicondyle of Humerus in residents of Jammu And Kashmir State. Paper read at 38th annual conference, 1989 of Anat. Soc. of India. Abstract published in Journal of Anatomical Society of India. 1990; 39:76.
5. Basu SK , Basu S. A contribution to the study of diaphysio-epiphysial relation at Knee of young Bengali girls. Ind J of Ped 1938; 5: 202-204.
6. Bokariya P. Effects of dietary habits on epiphyseal fusion. Journal of Ind. Academy of Forensic Med.- 2008/2009: page 34-37.
7. Cameron JAP. Estimation of age in Asiatic Girls, J Malaya. Br British Med Asso 1938; 12(1): 19-23.

8. Camps Francis E. Gradwohl's Legal Medicine 3rd ed. Bristol: John wright and Sons Ltd. 1976 p. 140-141.
9. Cardoso Hugo FV. Age estimation of adolescent young adult male and female skeletons II, Epiphysial Union at the Upper Limb and scapular Girdle in a modern Portuguese Skeletal sample. American J of Physical Anthropology, 2008, issue 137, page 97-105.
10. Chaurasia BD. Human Anatomy. Regional and applied 2nd edition.1989. CBS Publishers and Distributors. p. 3-11.
11. Davies, DA, Parson FG. The age order of the appearance and union of the normal epiphyses as seen by X-rays. J. Anat., 1927; 62:58-71.
12. Flecker H. Roentgenographic observations of the times of appearance of epiphysis. J Anat 1932; 67: 188-164.
13. Flecker H. Anatomical Society of India Vol.,67 October 1931.
14. Galstaun G. A study of ossification as observed in Indian subject. Ind j Med Res 1937; 25(1):267-324.
15. Garn SM, Rohman CG, Bluementhalt. Ossification sequence polymorphism and sexual dimorphism in skeletal development .Am J Physical Anthropology 1996: 101-115.
16. Gopalan C. Nutritional problems and programs in south East Asia, New Delhi: WHO Regional office for south East Asia; 1987.
17. Graham CB. Assessment of bone maturation methods and pitfalls. Radiol clin North America 1972; 10:185-202.
18. Haines RW, Mohiuddin A, Okpa FI and Viega-Pires JA. The study of early epiphyseal union in the limb girdles and majot long bones of man. J Anat.,1967, 101,4, page 823-831.
19. Hepworth SM. Determination of age in Indians from study of the calcification of the long bones. Ind Med Gaz 1929; 64:128.
20. Jit I, Kulkarni M. Time of appearance and fusion of epiphysis at medical end of clavicle. Indian J Med Res .1976 May: 64(5):773-82.
21. Knight, Bernard. Age Estimation: In, Forensic Pathology, Edward Arnold, A division of Hodder & Sttoughton London.1996 p. 109-114- 118.
22. Modi PJ. in chapter Personal Identity in Modi's Medical Jurisprudence and Toxicology, 22nd ed. edited by Mathiharan K and Patnaik AK. New Delhi: Butterworths India; 2005. p. 263 – 337.
23. Mehta HS. Age determination-Medical Law and Ethics in India. The Bombay Samachar Pvt. Ltd. Mumbai.1963;p.335-338 (cited in chapter Personal Identity in Modi's Medical Jurisprudence and Toxicology, 22nd ed. edited by Mathiharan K and Patnaik AK. New Delhi: Butterworths India; 2005. p. 263 – 337)
24. Reddy KSN. Identification-Growth in Individual bone, In the Essentials of Forensic Medicine and Toxicology. 29th ed. Hyderabad: K. Suguna Devi; 2009. p 64-71.

Original Article

A STUDY OF CORRELATION OF CHEILOSCOPY AND BLOOD GROUP

Dr. Prerna Gupta, Dr. Neeraj Gupta, Dr. BM Bannur, Dr. Anand B Mugadlimath,
Dr. Mandar Ramchandra Sane, Dr.Rekha Hiremath

Authors:

Dr. Prerna Gupta, Assistant Professor, Dept. of Anatomy, TRR Institute of medical sciences, Patancheru, Hyderabad.

Dr. Neeraj Gupta, Postgraduate student, Dept. of Forensic Medicine, BLDE, Shri B. M. Patil Medical college, Bijapur Karnataka.

Dr. BM Bannur, Professor and Head, Dept. of Anatomy, BLDE, Shri B. M. Patil Medical college, Bijapur Karnataka.

Dr. Anand B Mugadlimath, Associate Professor, Dept. of Forensic Medicine, Ashwini Rural Medical College Solapur Maharashtra.

Dr. Mandar Ramchandra Sane, Assistant Professor, Dept. of Forensic medicine, GMC Aurangabad Maharashtra.

Dr. Rekha Hiremath, Assistant Professor, Dept. of Anatomy, Ashwini Rural Medical College Solapur Maharashtra.

Number of Pages: Four

Number of Tables: Two

Number of Photographs: Nil

Address for correspondence: Dr. Prerna Gupta, Assistant Professor, Dept. of Anatomy,
TRR Institute of medical sciences, Patancheru, Hyderabad

Original Article

A STUDY OF CORRELATION OF CHEILOSCOPY AND BLOOD GROUP

Dr. Prerna Gupta, Dr. Neeraj Gupta, Dr. BM Bannur, Dr. Anand B Mugadlimath,
Dr. Mandar Ramchandra Sane, Dr. Rekha Hiremath

Abstract:

The lip prints are the normal lines and fissures in the zone of transition of human lip between mucosa and the skin. They are identifiable as early as the sixth week of intrauterine life. They are permanent, unchangeable even after death, and unique to each person except in monozygotic twins. The present study is being carried out to identify different types of lip prints their relation with blood groups and to see their uniqueness. Lip prints were obtained from 1st year medical students in the age group between 18 – 20 years. The lip prints were taken by applying lipstick and prints taken on bond paper. Prints were studied with the help of a magnifying lens by applying Suzuki's classification. The result and its implication will be discussed in the presentation.

Keywords: lip prints, personal identification, Suzuki's classification, Blood groups.

Introduction:

The lip prints are the normal lines and fissures in the zone of transition of human lip between mucosa and the skin.^{1,2} They are permanent, unchangeable even after death, and unique to each person except in monozygotic twins.¹⁻⁹

The analysis of fingerprints and bite marks are used to establish identity of an individual in the court of law. In the same way lip prints have also been considered to establish the identity of an individual especially in sexual assault cases. The blood alone is a very important entity in medicolegal practice for identification of an individual. Various blood group systems have been discovered. Amongst those, the ABO blood group system is the primary and most important because it is most common, conspicuous and easily detectable. The whole population of the world can be grouped into four groups based on ABO blood group system namely groups A, B, AB and O, further divided in to Rh positive and Rh negative depending on Rh antigen.

Objectives of study:

- To identify different patterns of lip prints and their relation with blood groups.
- To check the uniqueness of lip print.

Source of data:

The subjects under study were the first year medical students between age 18-20 yrs of BLDE University's Shri B.M. Patil Medical College, hospital and research centre, Bijapur.

Method of data collection:

The study was conducted over a period of 2 months. First year medical students of Indian origin, belonging to age group of 18–20 years were taken as subjects. Written informed consent of the subjects were taken. Lipstick was applied on the lips of the subject with a single stroke. Then with the help of a paper, the centre portion of lips was dabbed first and then left and right corners of lips pressed, applying uniform pressure, taking care to avoid sliding of lips to prevent smudging of the print. After the lip prints were acquired, details such as name, sex, age, blood group was documented. Each lip print was assigned a serial number.

Each lip print was compared manually with others using a magnifying glass to test the uniqueness of lip prints.²⁻⁴. Patterns of lip print were studied by applying Suzuki's classification.¹⁰⁻¹¹

Ethical clearance--Has been obtained from the ethical committee of the institution

Inclusion criteria:

Age between 18 -20 years of both males and females.
Subjects free from any active or passive lesions on their lips.

Exclusion criteria:

Students with known hypersensitivity to lip sticks.
Active or passive lip lesions and non-resident Indians.
Student who do not give consent.

Sample size:

1. Males who gave consent = 60
2. Females who gave consent =68
3. Total sample size = 128

Type of study:

Prospective study of duration two months and data was analysed by using following statistical methods.

- Tabulated presentation.
- Mean +/- Standard deviation.
- Chi-square

Suzuki's classification:

- Type I: Vertical, complete (end-to-end) longitudinal fissures.
- Type I' (one - dash): Incomplete vertical longitudinal fissures.
- Type II: Branching, Y-shaped pattern.
- Type III: Criss-cross pattern.
- Type IV: Reticular, typical chequered pattern, fence like

Results:

The chi-square was applied to test whether there was any association between the type of lip print and blood group of the subjects.

Table 1. Percentage of lip print types in males and females

Classification of lip prints	Male students		Female students		Total students	
	240 Quadrants	%	272 Quadrants	%	512 Quadrants	%
Type I	190	79.17	172	63.78	362	70.70
Type I'	75	31.25	81	29.78	156	30.47
Type II	64	26.67	74	27.20	138	26.95
Type III	67	27.92	89	32.72	156	30.47
Type IV	51	21.25	71	26.10	122	23.83

In males frequencies of lip prints: TYPE I > TYPE I' >TYPE III >TYPE II > TYPE IV
In females frequencies of lip prints: TYPE I > TYPE III > TYPE I' >TYPE II> TYPE IV

Table 2 : Correlation of lip prints with blood groups cross tabulation.

Blood Group	Types of lip prints					Total
	Type I	Type I'	Type II	Type III	Type IV	
A ^{+ve}	1 (2.63%)	1 (3.84%)	5 (20%)	3 (11.11%)	0	10 (7.81%)
A ^{-ve}	1 (2.63%)	0	0	0	1 (8.33%)	2 (1.56%)
B ^{+ve}	6 (15.78%)	5 (19.23%)	1 (4%)	15 (55.56%)	3 (25%)	30 (23.43%)
B ^{-ve}	0	0	1 (4%)	0	0	1 (0.78%)
O ^{+ve}	25 (65.78%)	10 (38.46%)	15 (60%)	8 (29.6%)	7 (58.3%)	65 (50.78%)
O ^{-ve}	0	0	0	0	0	0
AB ^{+ve}	5 (13.15%)	10 (38.46%)	3 (12%)	1 (3.7%)	1 (8.33%)	20 (15.62%)
AB ^{-ve}	0	0	0	0	0	0
Total	38	26	25	27	12	128

Discussion:

It was observed that Type I was the most frequently observed in both the sexes. Other works on Indian subjects have yielded varying results. Vahanwalla and Parekh in their study in Mumbai found that Type I was the most frequent.⁴ Sivapathasundharam, Prakash and Sivakumar studied the lip prints of Indo-Dravidian population and noted that Type III was predominant.² PrateekRastogi in his study among medical students found that the incidence of Type I was the highest among males which is similar to our finding.¹²

Conclusion:

Cheiloscopy is a relatively new field among the large number of identification tools available to the forensic expert. The present study compared the types of lip prints with the ABO and Rh blood groups of the subjects. It was found that there was no correlation in the total subject population. Therefore we should not predict the blood group by her/his lips print. Lip prints are unique to an individual and can be used to fix the identity of a person; that they remain stable over time and that lip prints show gender differences.

Uniqueness of lip prints:

- Lip prints are unique to individuals and remain unchanged throughout life. Identifiable lip prints can be obtained up to 30 days after being produced.
- Lipstick smears are frequently encountered in forensic science laboratories as one important form of transfer evidence. Smears can also be found in other places, such as glasses, cups, spoons or cigarette butts, therefore indicating some kind of relationship between a suspect and the crime scene.¹³
- Cheiloscopy is still an inexact science and more studies need to be done to confirm its validity.

Limitations of Cheiloscopy:

The lip print is produced by a substantially mobile portion of the lip. This fact alone explains the reason why the same person can produce different lip prints, according to the pressure, direction and method used in taking the print.

If lipstick is used, the amount can also affect the print. Smudging of lip prints is one of the major limitations of using lip sticks as in the presents study.

Manual register of the overlay is another problem, due to the possibility of some subjectivity.

Another factor to be considered is the existence of some pathological conditions (lymphangiomas, congenital lip fistula, lip sclerodermi, Merkelson–Rosenthal syndrome, syphilis, lip cheilitis among others), which can invalidate the cheiloscopic study.

References:

1. Caldas IM, Magalhaes T, Afonso A. Establishing identity using cheiloscopy and palatoscopy. *Forensic Sci Int.* 2007;167:1-9.
2. Sivapathasundharam B, Prakash PA, Sivakumar G. Lip Prints (Cheiloscopy). *Indian J Dent Res.* 2001; 12(4):234-37.
3. Tsuchihashi Y. Studies on Personal Identification by Means of Lip Prints. *Forensic Sci.* 1974; 3: 233-48
4. Vahanwalla SP, Parekh BK. Study on Lip Prints as an Aid to Forensic Methodology. *J Forensic Med and Toxicol.* 2000; 17(1): 12-18.
5. Vahanwala SP, Nayak CD, Pagare SS. Study of lip prints as aid for sex determination. *Medico-Legal Update.* 2005;5(3): 93-98.
6. Hirth L, Gottsche H, Goedde HW. Lip print- variability and genetics. *Humangenetics.* 1975;30(1):47-62.
7. Coward RC. The stability of lip print characteristics over time. *J Forensic Odontostomatol.*2007;25(2): 40–56.
8. Utsuno H, Kanoh T, Tadokoro O, Inoue K. Preliminary study of PM identification using lip prints. *Forensic Sci Int.* 2005;149:129-132.
9. Kasprazak J. Possibilities of cheiloscopy. *Forensic Sci Int.* 1990;46:145-151.
10. Suzuki K, Tsuchihashi Y. A new attempt of personal identification by means of lip prints. *Can Soc Forensic Sci.* 1971;4:154-158.
11. Jaishankar S, Jaishankar N, Shanmugam S. Lip prints in personal identification. *J Indian Acad Dent Specialists.* 2010;1(4):23-26.
12. Rastogi P and PridaA. Lip prints – an aid in identification .*Australian Journal of Forensic sciences.*2011;1-8, i First article.
13. Ball J. The Current Status of Lip Prints and Their Use for Identification. *J Forensic Odontostomatol.* 2002; 20: 43-46.

Acknowledgement:

Authors acknowledge the help received from the scholars whose articles are cited and included in references of this manuscript. The authors are also grateful to authors / editors /publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed.

Original article

VIOLENT ASPHYXIAL DEATH DUE TO HANGING: A PROSPECTIVE STUDY

Dr. SH Bhosle, Dr. AK Batra, Dr. SV Kuchewar

Authors:

Dr. Santosh H. Bhosle. MBBS, MD, Assistant Professor, Department of Forensic Medicine, Dr. S.C. Govt. Medical College, Nanded. (Maharashtra).

Dr. Anil K. Batra. MBBS, MD, LLB, Professor & Head, Department of Forensic Medicine, Govt. Medical College, Akola. (Maharashtra).

Dr. Sharad V. Kuchewar. MBBS, MD, Assistant Professor, Department of Forensic Medicine, Shri V. N. Govt. Medical College, Yavatmal. (Maharashtra).

Number of pages: Seven

Number of Tables: Five

Number of Charts: Two

Address for correspondence:

Dr. Santosh Harishchandra Bhosle
Assistant Professor, Department of Forensic Medicine,
Dr. S.C. Govt. Medical College, Nanded. Pin-431601
e-mail: santoshbhosle09@gmail.com

Original article

VIOLENT ASPHYXIAL DEATH DUE TO HANGING: A PROSPECTIVE STUDY

Dr. SH Bhosle, Dr. AK Batra, Dr. SV Kuchewar

Abstract:

Hanging is one of the common methods of committing suicide and it is considered suicidal unless contrary is proved. In the present study total 84 cases of asphyxial death due to hanging were studied during October 2008 to June 2010 in Department of Forensic Medicine and Toxicology, at Shri Vasantrao Naik Government Medical College, Yavatmal (Maharashtra).

In our study, almost all cases (98.81%) of hanging were suicidal in nature. There was male preponderance (78.57%) and age group 21-40 years (61.90 %) was the most vulnerable for committing suicide by hanging. Most of victims of suicidal hanging were farmers (30.12%) and laborers (24.10%). The most common place of committing hanging was home (69.88%) and most common ligature material used for suicidal hanging was the nylon rope (53.01%). Complete ligature mark was found in significant number of cases of hanging with running noose and throat skeleton fracture were more common with complete hanging and with increasing age.

Key words: Hanging, farmer suicide, nylon rope, throat skeleton fracture.

Introduction:

The term 'asphyxia' literally means lack of oxygen, though etymologically, asphyxia means 'absence of pulsations'¹. The usual relevance of asphyxial death in Forensic context is violent asphyxia. In violent asphyxial deaths, exchange of air between atmosphere and lung beds is prevented by some violent mechanical means. Usual causes of violent asphyxial deaths are hanging or strangulation, choking and drowning.

Hanging is a form of ligature strangulation in which the force applied to the neck is derived from a gravitational drag of the weight of the body or part of the body¹. Apart from now rare 'lynching', hanging is almost always suicidal or accidental, the former being by far most common. Occasional cases of homicidal hanging have been reported. Examples have also occurred of people killed in some other way and later suspended to simulate hanging². Hence death due to hanging is one of the most complex and controversial areas of asphyxial deaths. To ascertain cause and manner of death in cases of hanging, meticulous examination of ligature mark, neck structure findings and other autopsy findings are much helpful². Also visit to the scene of crime is very much helpful to ascertain circumstances and manner of hanging.

Yavatmal is one of those districts of Maharashtra in which majority of population are engaged in agricultural work. Farmer suicides are very common in this region and hanging being one of the common methods used by farmers to commit suicide. The present study was carried out to know demographic features, characteristics of ligature mark, its relationship with the knot & injuries to neck structures in deaths due to hanging and to emphasize importance of meticulous examination of neck in such cases.

Material & Methods:

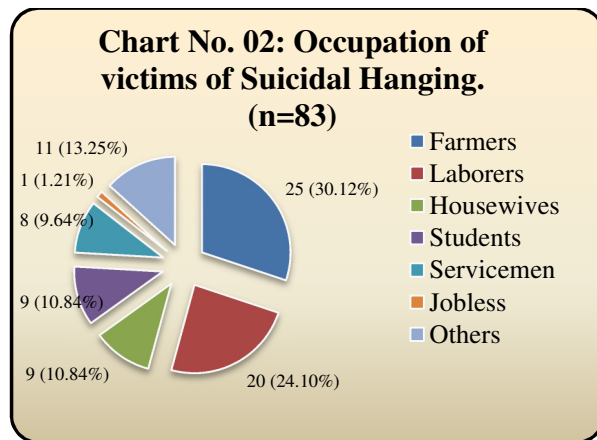
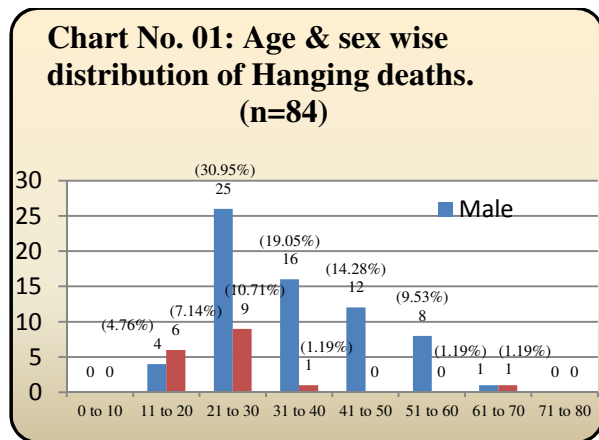
The present study was conducted in Department of Forensic Medicine and Toxicology, at Shri Vasantrao Naik Government Medical College, Yavatmal (Maharashtra). Total 84 cases of

asphyxial death due to hanging were studied during October 2008 to June 2010. The details about the victims regarding the age, sex, circumstances of death, type of ligature material, manner and supposed cause of death were obtained from police reports and relatives of deceased.

The meticulous examination of ligature material, whenever available, was carried out in this study. A thorough and complete autopsy was carried out in each case with special reference to injuries to neck structures. The data of 84 cases of death due to hanging were recorded, compiled and analyzed statistically.

Results:

During study period, 84 cases of death due to hanging were observed. Out of 84 cases, most of the cases were suicidal in nature (98.81%). Only one case of homicidal hanging was observed during study period and victim was female. Accidental hanging was not observed during study period.



Majority of deaths due to hanging were males (78.57%) with male: female ratio 1:0.27. The highest number of cases of death due to hanging were in the age group of 21-30 years (41.67%) followed by age group 31-40 years (20.24%)(Chart 01). The analysis of the occupations of victims who died due to suicidal hanging revealed that, hanging was more common in farmers (30.12%) and laborers (24.10%) followed by housewives (10.84%), students (10.84%), and servicemen (09.64 %) (Chart 02).

Table No. 01: Place of suicidal hanging. (n=83)

Place of occurrence	Total (%)
Home	58 (69.88)
Farm	11 (13.25)
Public Places	04 (04.82)
Forest	06 (07.23)
Other Places	04 (04.82)
Total	83 (100)

The most common place of committing suicide by hanging was home (69.88%) followed by farm (13.25%) and forest (07.23%) (Table 01).

The ligature material most commonly used to commit suicide by hanging was nylon rope (53.01%) followed by long handkerchief (06.03%) and Chunni (06.03 %). Jute rope, cotton rope and sari were used as ligature material in 03.61% cases each while the ligature material could not be

ascertained in 16 (19.28%) cases (Table 02).

Table No. 02: Ligature material used for Suicidal hanging. (n=83)

Ligature material	Number	%
Nylon rope	44	53.01
Long handkerchief	05	06.03
Chunni	05	06.03
Jute rope	03	03.61
Cotton material	03	03.61
Sari	03	03.61
Coconut rope	01	01.19
Shawl	01	01.21
Towel	01	01.21
Electric wire	01	01.21
Not Ascertained	16	19.28
Total	83	100

Table No. 03: Relation of type of ligature mark with type of knot. (n=84)

Ligature knot	Ligature mark		Total
	Complete	Incomplete	
Fixed	12 (40.00%)	18 (60.00%)	30 (100%)
Running	15 (51.72%)	14 (48.22%)	29 (100%)
Not known	05 (20.00%)	20 (80.00%)	25 (100%)

The fixed noose was observed in 30 (35.71%) cases; out of which 12 (40%) cases showed complete ligature mark and 18 (60%) cases showed incomplete ligature mark around neck. The running noose was seen in 29 (34.52%) cases; of which ligature mark completely encircling around neck was observed in 15 (51.72%) cases (Table 03).

Table No. 04: Particulars of ligature mark in hanging. (n=84)

Particulars of Ligature mark		Number	%
Type	Complete	32	38.10
	Incomplete	52	61.90
Level	Above level of thyroid	70	83.33
	At level of thyroid	13	15.47
	Below level of thyroid	01	01.19
Direction	Oblique	83	98.81
	Horizontal	01	01.19
Depth	Deep	65	77.38
	Shallow	19	22.62

In 52 (61.90%) cases, the ligature mark was incomplete and in 32 (38.10%) cases, ligature mark was completely encircling neck. The ligature mark was observed above the level of thyroid cartilage in 70 (83.33%) cases, at the level of thyroid cartilage in 13 (15.47%) cases and below thyroid cartilage in 01 (01.19%) case. In 83 (98.81%) cases, the ligature mark was passing obliquely upward and ligature mark was horizontal in only 01 (01.19%) case. Ligature mark was grooved in 65 (77.38%) cases and shallow in 19 (22.38%) cases (Table 04).

Table No. 05: Throat skeleton fracture in relation to type of suspension. (n=84)

Throat skeleton fracture	Type of suspension		
	Complete (%)	Partial (%)	Not known (%)
Fracture of Hyoid bone	07 (08.33)	03 (03.57)	03 (03.57)
Fracture of Thyroid cartilage	01 (01.19)	01 (01.19)	00 (00)
Fracture of hyoid & Thyroid	00 (00)	00 (00)	00 (00)
No fracture	26 (30.95)	28 (33.33)	15 (17.85)
Total	34 (100)	32 (100)	18 (100)

In 34 (40.47%) cases complete suspension of body was noted and 32 (38.09%) cases there was partial suspension. The throat skeleton fracture was observed in 07 (20.58%) cases of complete suspension and 03 (9.37%) cases of partial suspension (Table 05).

Out of 22 cases above 40 years of age, throat skeleton fracture was observed in 08 (36.37%) cases while out of 62 cases below 40 years of age 07 (11.29%) cases showed throat skeleton fracture.

Discussion:

Hanging is one of the common methods of committing suicide and it is considered suicidal unless contrary is proved. In our study, 83 (98.81 %) cases of hanging were suicidal and 01 (01.19%) case of homicidal hanging was observed. Similar findings were reported by Naik et al³ (98.44%), Batra et al⁴ (100%), Azmak D⁵ (100%), Simonsen J⁶ (96.25%), Davidson et al⁷ (95.5%), Bowen DA⁸ (93.53%), Cooke et al⁹ (93.21%), and Elfawal et al¹⁰ (96.7%). Single homicidal hanging noted in our study was of dyadic death where newly married female was hanged by her husband and later he committed suicide by hanging. Similar type of homicidal hanging in dyadic death was reported by Lew EO¹¹.

In this study, majority of hanging deaths was contributed by males (78.57%). Similar finding with male preponderance was observed by Elfawal et al¹⁰ (80.32%), Nikolic et al¹² (76%), Paparo et al¹³ (75.62%), Dixit et al¹⁴ (75%), Bowen DA⁸ (82.98%), Batra et al⁴ (83.76%), Luke et al¹⁵ (83.60%), Morild I¹⁶ (73%), Sharma et al¹⁷ (66.66%), Singh et al¹⁸ (66.55%), Simonsen J⁶ (60%), Azmak D⁵ (83.9%), Uzun et al¹⁹ and Sharma et al²⁰. This does not mean that males commit suicide more than females. It only signifies that, men prefer to commit suicide by hanging than women who prefer to commit suicide by other means like burning, drowning and poisoning⁴. Naik et al³ reported female preponderance (62.2%) which is in contrast with our study.

The study showed that 21-40 years (61.90%) was the most vulnerable age period for committing suicide by hanging. High incidence of hanging in middle age group and minimal incidence in extremes of age was also reported by many researchers^{4,5,10,14-19,21} with varying percentage. The age group of 21-40 years is the most active phase in life wherein exposure to anxiety, stress, strain and various adverse circumstances occur. Economic Problems, unemployment, failure in love, alcohol addiction, and emotional instability were the alleged reasons for committing suicide in this age group. Hanging more common in higher age group than our study was reported by Bowen DA⁸ and Simonsen J⁶.

In our study, analysis of the occupations of victim who committed suicidal hanging revealed that farmers (30.12%) and laborers (24.10%) constitute major group. The study of Elfawal et al¹⁰ reported that the most of the victims of suicidal hanging were from low socio-economic class namely laborers and domestic workers. The present study was carried out in district of Vidarbha region of Maharashtra where farmers commit suicide by various means due to failure of crops, poverty, bankruptcy, alcohol addiction, and stress & strain.

In present study the most common place of hanging was home in 69.88%. Similar observations were reported by Cooke et al⁹ (71%), Uzun et al¹⁹ (83.33%), Elfawal et al¹⁰ (95.08%) and Bowen DA⁸. Usually person prefers any secluded place which suit for his/her purpose. The victim being very well aware of the home and its surroundings, it suits his or her needs for hanging. The second most commonly preferred secluded place for committing suicide observed in our study was farm and other places were forest, public ground, garden, school, shop and Hindu cemetery.

Nylon rope was the most common ligature material (53.01%) used for hanging in our study. Similar finding was also reported by Sheikh et al²² (53.02%). Dixit et al¹⁴, Uzun et al¹⁹ & Cooke et al⁹ reported rope as most common ligature material. Elfawal et al¹⁰ reported that popular plastic clothesline (rope) (85.41%) was most common ligature material used for hanging. Nylon rope was the commonest ligature material used for hanging in our study was probably because most of the victims of hanging were farmers, farm workers or most of them belong to agricultural family background. Nylon rope is easily available due to common use for various purposes by farmers & for domestic uses and also, as it is cheap, the incidence of its use in suicidal hanging was more. Sharma et al¹⁷ reported that common ligature material was Chunni (30.90%) which is in contrast with our finding. The ligature material used by the victim for hanging may be anything available at that moment, which includes any household article or belongings of the victim. This view is further strengthened by the findings in our study which showed that other ligature material used for hanging were long handkerchief, chunni, sari, shawl, towel, jute rope, cotton rope, coconut rope and electric wire.

The complete ligature mark around neck was observed in 51.17% of running noose cases and 40% of fixed noose cases. The ligature mark completely encircling the neck is usually found with running type of noose because the noose moves towards the neck due to weight of body and thus completely constricting the neck; giving complete ligature mark.²³ However in present study, complete ligature mark around neck was observed significantly even in cases with fixed noose (40%). This could be due to the nylon rope being very smooth & slippery in nature, and during hanging the knot usually slips towards the neck giving complete ligature mark.

In the present study ligature mark was completely encircling the neck in 32 (38.10%) cases and was incomplete in 52 cases (61.90%). The ligature mark was commonly observed above the level of thyroid cartilage in hanging (83.33%). The similar findings were reported by Sharma et al²⁰ (84.62%) and Mukherjee JB²³ (80%). Lower incidence ligature mark above level of thyroid cartilage was reported by Elfawal et al¹⁰ (65.57%) and Dixit et al¹⁴ (77%). Ligature mark below the level of thyroid cartilage in significant percent was reported by Davidson et al⁷ (6.66%), Elfawal et al¹⁰ (24.59%) and Dixit et al¹⁴ (23%). The high reporting may be because that, they had not differentiated mark at the level of thyroid cartilage or below it; but clubbed both as below the level of thyroid cartilage. In our study we observed obliquely placed ligature mark in 83 (98.81%) cases. Similar finding was reported by Sharma et al¹⁷ (97.8%). Horizontal ligature mark situated below the level of thyroid cartilage was observed in only one case (01.19%) with partial hanging in almost lying down position where only head, neck and upper chest was not touching ground. In the present case, the victim had consumed alcohol prior to committing suicide. Deep, grooved mark was observed in 65 (77.38%) cases. In our study as most common ligature material used for hanging was nylon rope, grooved ligature mark was observed more commonly.

The present study showed fractures of hyoid bone in 13 (15.47%) cases, which is similar to finding of Dixit et al¹⁴ (14.38%) and Morild I¹⁶ (16.00%). The fracture of hyoid bone noted by different authors^{5,6,15,22} varies with different percent. The fractures of thyroid cartilage were noted in 02 (2.38%) cases in present study correlating with Feigin G²⁴ (06.40%) but it does not correlate with Morild I¹⁶ (12.50%), Luke et al¹⁵ (13.11%) and Dixit et al¹⁴ (15.60%). Paparo et al¹³ noted throat skeleton fracture in 20% cases and Feigin G²⁴ in 9% cases. Elfawal et al¹⁰, Bowen DA⁸, Naik et al³ did not find any fracture of hyoid bone or thyroid cartilage in their study. The age composition of study group, type of ligature material used and type of hanging

probably plays the role in sustaining the neck structure injuries and these things differ from one study to other. Thus, it is expected to vary from one study to another.

In 23.5% cases of complete hanging and 12.5% of partial hanging cases the throat skeleton was fractured in our study. Luke et al¹⁵ (25.8%) and Morild I¹⁶ (26.89%) reported similar finding of more common fracture of throat skeleton with complete hanging. Higher percentage of throat skeleton fracture with complete suspension of body (56.66%) than partial suspension (38%) was noted by Simonsen J⁶. The higher percentage of fracture in completely suspended bodies resulted from rapid and forceful constriction of the neck, the ligature supporting the entire body weight and the more force acting on the throat skeleton.

The frequency of throat skeleton fracture in hanging was more with increasing age of the victim. Similar finding were reported by Luke et al¹⁵, Davidson et al⁷, Morild I¹⁶ and Nikolic et al¹². All these authors described that with advancing age the bones and cartilages of the neck get calcified, become rigid and brittle, thus more liable to fracture.

Conclusion:

Hanging being viewed as giving swift painless death and with easily available ligature material & ligature points in secluded place without arousing much suspicion, this mode is increasingly adopted to commit suicide. Peak incidence of mortality due to hanging is in young age groups (21-40 years). The present study throws some light on the emerging trends that nylon rope is increasingly used for committing suicidal hanging. More incidence of throat skeleton fracture is observed with complete hanging & with increasing age.

References:

1. Saukko P and Knight B. Knight's Forensic Pathology. Third Ed. London: Arnold Publishers; 2004. p. 352-394.
2. Camps FE. Gradwohl's Legal Medicine. Third Ed. Bristol: John Wright & Sons LTD; 1976. p. 326-336.
3. Naik S, Patil DY. Fracture of hyoid bone in cases of asphyxial deaths resulting from constricting force around the neck. Journal of Indian Academy of Forensic Medicine 2005; 27(3); 149-153.
4. Batra AK, Dongre AP. A Preliminary Analysis of Medico-legal Autopsies Performed over Five years in a rural Health District of Maharashtra state of India. Journal of Forensic Medicine and Toxicology 2003 Jan – June; 20(1): 41-46.
5. Azmak D. Asphyxial deaths: a retrospective study and review of literature. American Journal of Forensic Medicine and Pathology 2006 Jun; 27(2):134-144.
6. Simonsen J. Patho-anatomic findings in neck structures in asphyxiation due to hanging: A survey of 80 cases. Forensic Science International 1988; 38: 83-91.
7. Davidson A, Marshall TK. Hanging in Northern Ireland-a survey. Med Sci Law 1986; 26: 23-28.
8. Bowen DA. Hanging – a review. Journal of Forensic Science international 1982 Nov-Dec; 20(3): 247-9.
9. Cooke CT, Cadden GA, Margolius KA. Death by hanging in Western Australia. Pathology 1995 Jul; 27(3): 268-72.
10. Elfawal MA, Awad OA. Death from hanging in the eastern province of Saudi Arabia. Med Sci Law 1994 Oct; 34(4): 307-12.

11. Lew EO. Homicidal hanging in a dyadic death. *Am J Forensic Med Pathology* 1988; 9: 283-386.
12. Nikolic S, Mistic J, Atanasijevic T, Djokic V, Djonic D. Analysis of neck injuries in hanging. *Am J Forensic Med Pathol* 2003 June; 24(2): 179-82.
13. Paparo PG, Siegel H. Neck Markings and fractures in suicidal hanging. *Forensic Science International* 1984 Jan; 24(1): 27-35.
14. Dixit PG, Mohite PM, Ambade VN. Study of histopathological changes in thyroid, salivary gland and lymph nodes in hanging, *Journal of forensic medicine and toxicology*. 2001 July-Dec; 18(2): 1-4.
15. Luke JL, Reay DT, Eisele JW, Bonnell HJ. Correlation of circumstances with pathological findings in asphyxial deaths by hanging: a prospective study of 61 cases from Seattle, WA. *Journal of Forensic Sciences* 1985 Oct; 4: 1140-47.
16. Morild I. Fractures of neck structures in suicidal hanging. *Med Sci Law* 1996 Jan; 36(1): 80-4.
17. Sharma BR, Harish D, Singh VP, Singh J. Ligature Mark on the neck: how Informative? *Journal of Indian Academy of Forensic Medicine* 2005; 27(1): 10-15.
18. Singh P, Marak F, Longkumer K, Momonchand A. Suicides in Imphal. *Journal of Indian Association of Forensic Medicine* 2005; 27(2): 85-86.
19. Uzun I, Buyuk Y, Gurpinar K. Suicidal hanging: fatalities in Istanbul retrospective analysis of 761 autopsy cases. *J Forensic Leg Med* 2007 Oct; 14(7): 406-9.
20. Sharma BR, Harish D, Anup Sharma, Sharma S, Singh H. Injuries to neck structures in death due to constriction of neck, with special reference to hanging. *Journal of Forensic and Legal Medicine* 2008 July; 15(5): 298-305.
21. Ambade VN, Godbole HV, Kukde HG. Suicidal and homicidal deaths: a comparative and circumstantial approach. *J Forensic Leg Med*. 2007 Jul; 14(5): 253-60.
22. Sheikh MI, Agarwal SS. Medicolegal implications of hyoid bone fracture – A study. *Journal of Indian Academy of Forensic Medicine* 2001 Apr-Jun; 23(4): 61-63.
23. Mukharjee JB. *Forensic Medicine and Toxicology*. R N Karmakar editor. Third edition New Delhi: Arnold Associates; 2007. p. 571-617.
24. Feigin G. Frequency of neck organ fractures in hanging. *American J Forensic Med Pathol*. 1999 Jun; 20(2):128-30.

Case Report

ARTEFACT OR CAUSE OF DEATH: CRITICAL ANALYSIS OF AUTOPSY FINDINGS TO RESOLVE THE DILEMMAS – A CASE REPORT.

Dr. HS Tatiya, Dr. SB Punpale, Dr. AA Taware, Dr. VT Jadhav, Dr. AL Bandgar

Authors

Dr. H. S. Tatiya, JR- II, Department of Forensic Medicine and Toxicology, B.J. Govt. Medical College and S.G. Hospitals Pune.

Dr. S. B. Punpale, Professor and Head, Department of Forensic Medicine and Toxicology, B.J. Govt. Medical College and S.G. Hospitals Pune.

Dr. A. A. Taware, Associate Professor, Department of Forensic Medicine and Toxicology, B.J. Govt. Medical College and S.G. Hospitals Pune.

Dr. V. T. Jadhav, Assistant Professor, Department of Forensic Medicine and Toxicology, B.J. Govt. Medical College and S.G. Hospitals Pune.

Dr. A. L. Bandgar, JR- I, Department of Forensic Medicine and Toxicology, B.J. Govt. Medical College and S.G. Hospitals Pune.

Number of Pages: Five

Number of Tables: Nil

Number of Graph: Nil

Number of Photographs: Two

Corresponding Author: Dr. H. S. Tatiya
Department of Forensic Medicine and Toxicology,
B.J. Govt. Medical College and S.G. Hospitals Pune, 411 001.
9422789579, hstatiyakhane7@gmail.com

Case Report

ARTEFACT OR CAUSE OF DEATH: CRITICAL ANALYSIS OF AUTOPSY FINDINGS TO RESOLVE THE DILEMMAS – A CASE REPORT.

Dr. HS Tatiya, Dr. SB Punpale, Dr. AA Taware, Dr. VT Jadhav, Dr. AL Bandgar

Abstract

Regurgitation and aspiration of Gastric contents in the respiratory tract is a common agonal artefact seen in forensic practice. This situation is commonly found at postmortem, if victim is unconscious or under the influence of alcohol, drug or anesthesia or during a fit of epilepsy and in dead bodies that have started decomposing. Quite infrequently it may be found in fresh bodies that have undergone sudden unexpected and unattended death leading to a dilemma as to the real cause of death in absence of any other substantial evidence. The real challenge for the autopsy surgeon is to differentiate between ante-mortem aspiration and postmortem spill of gastric contents into the respiratory tract, a well-known artefact in such unattended deaths. Misinterpretation of such artefacts can lead to wrong diagnosis of cause of death. In present case, the death was sudden and unattended and gastric contents in the respiratory tract led to a dilemma as to the real cause of death. Dilemmas of the case with difficulties in diagnosis and critical analysis of autopsy findings to resolve the dilemmas are being presented herewith.

Key Words: Ante-mortem aspiration, post mortem spill, critical analysis, dilemmas.

Introduction

Sudden death can be both natural and unnatural. In sudden natural deaths, the immediate cause of death is usually found in the cardiovascular system [45-50%] followed by the respiratory system [10-15%] ^[1]. In the respiratory system, choking from mechanical obstruction is one of the causes for sudden and unexplained deaths. This mechanical obstruction is common due to foreign bodies, food bolus, hemorrhages or acute obstructive lesions. Regurgitated stomach contents resulting into choking is a not a common entity and literature available on death from such choking is also not forthcoming ^[2]. On the other hand regurgitation and aspiration of gastric contents is a common agonal artefact ^[3]. In cases of sudden death, finding of gastric contents in air passages is by no means as significant as the presence of freshly swallowed food. Gastric contents are commonly found in the larynx, trachea and bronchi at autopsy when no other evidence of aspiration exists and when there is a clear and unconnected cause of death. Gastric contents may reach the air passages from spontaneous agonal regurgitation or during pumping of chest and abdomen during resuscitation attempts. This makes the finding of gastric contents in the respiratory tract less significant ^[4]. The finding of small amounts of food material in the airway at autopsy does not indicate that the individual choked to death. One can attribute a death to aspiration only if the air passage below the level of larynx is completely occluded by food. It is rarely seen in medico-legal autopsies and is most common in patients who have impaired functioning of central nervous system ^[5]. A large proportion of deaths from choking occur before any possible hypoxic manifestations have time to take effect. These fatalities might be attributed to cardiac arrest, either purely neurogenic or accelerated by excess catecholamine release from the adrenaline response. Aspiration of vomit, as a cause of death, must be used with great caution unless there is an antemortem medical witness to it. The major exception, however, is acute alcohol intoxication, where if copious inhalation of stomach contents right down to the secondary bronchi is confirmed, then in the absence of significant natural

disease, injury or other toxicity, choking associated with a high blood alcohol level may reasonably be incriminated as the cause. However, it is not an autopsy diagnosis to be made lightly^[4].

Case Report

A dead body of a 56-year-old male, farmer by occupation was brought for postmortem examination to the Forensic Medicine Department, B.J. Government Medical College and S. G. Hospitals, Pune. The deceased was brought dead to the casualty of the hospital. History revealed that the deceased had an afternoon meal at around 2.00 pm and went on work at his farm for plucking of grass weeds, where at around 3.30pm he was found in unconscious state by the trace passers and was rushed to the hospital at around 4:30 pm; where the duty doctor declared him as brought dead. On inquiring about the deceased there was no past history of any major illness. There was no history of previous episodes of unconsciousness or being on any medication. He had taken medicines long back for a repetitive complaint of epigastric pain with on and off bouts of regurgitation and retrosternal chest pain after meals.

Postmortem examination was conducted on the same day. External examination was essentially negative with no significant finding. Rigor mortis was well marked in neck and upper limbs of body and lividity was present at back and buttocks, purple red colored and was fixed. Internal examination showed congested viscera. Heart did not show any sign of recent ischemia and coronaries were grossly patent. The yellowish green semi digested semisolid food material embedded in mucous secretions was present in the larynx and trachea up to tracheal bifurcation. Also there was evidence of yellowish green fluid lining the mucosa of whole respiratory tract. Similar food material and fluid was present in the stomach (Ref to photographs 1 and 2 below).



Photo 1: The yellowish green semi digested semisolid food material embedded in mucous secretions at tracheal bifurcation with evidence of yellowish green fluid lining the mucosa.

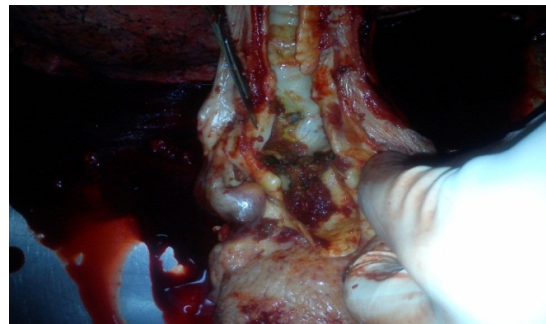


Photo 2: The yellowish green semi digested semisolid food material present in the larynx.

Viscera were sent for chemical analysis to rule out poisoning and alcohol intoxication and also tissues from the organs were preserved for histopathology. Opinion regarding the cause of death was reserved till the pending reports of chemical analysis and histopathological examination.

The Chemical laboratory investigations were negative for alcohol and poison. Histopathology of heart was unremarkable. There was no evidence of recent or old myocardial infarction. However histopathology of lung pieces showed presence of vegetable matter in the terminal bronchioles as well as in some alveoli. There was also evidence of minimal leucocyte clustering around the foci of vegetable matter in the bronchioles as well as

the alveoli. At many places, the alveoli were expanded with broken septa. After going through the case details available few dilemmas occurred in our minds as:

1. Whether the food material found in the respiratory tract was ante mortem phenomenon or postmortem spill?
2. Whether such autopsies should be labeled as negative autopsies or a questionable cause of death i.e. choking can be given as the cause of death?
3. If choking is the cause of death, what is the mechanism of death?
4. What is the reason of aspiration of gastric contents in the respiratory tract?

A good amount of literature was searched and after analyzing the significant microscopic findings associated with the autopsy findings and history of the case in backdrop of literature the opinion as to the cause of death was finalized as -“**Choking from aspiration of gastric contents**”.

Discussion

In forensic pathology, an artefact is any change caused or a feature introduced into body after death that is likely to lead to misinterpretation of medicolegally significant antemortem findings. It is the duty of the autopsy pathologist to interpret artefacts correctly. The misinterpretation can lead to wrong cause and manner of death^[6]. After going through the literature available we deduced the answers to our queries as follows:

1. Whether the food material found in the respiratory tract was ante mortem phenomenon or postmortem spill?

Regurgitation and aspiration of gastric contents is a common agonal artefact. It may be seen in natural deaths, as a terminal event, or due to handling of body or due to resuscitation^[3]. Gastric contents in respiratory tract are commonly found at postmortem in acute alcoholism, occasionally during epileptic fit, and in dead bodies that have started decomposing^[2]. According to Saukko and Knight, there is no reliable method of distinguishing agonal or even early postmortem over-spill from true vital aspiration unless clinical or other witnessed evidence is available^[4]. On the other hand, according to Modi, vomited matter may regurgitate into the larynx and by inspiratory efforts may be aspirated into the smaller bronchioles so as to result in suffocation. If there is postmortem spill, these contents cannot reach the smaller bronchi and bronchioles^[7]. In the present case, no positive autopsy finding was observed except for the presence of yellowish green semi digested semisolid food material embedded in mucous, present in the larynx and trachea up to tracheal bifurcation with evidence of yellowish green fluid lining the mucosa of whole respiratory tract, similar to that present in the stomach. This was an unusual finding considering the fact that the deceased was not suffering from epilepsy, chemical analysis did not reveal alcohol, there was no any resuscitation and decomposition had not set in [postmortem was done within 6-8 hours of death], so it cannot be easily overlooked as a postmortem artefact. In the present case, grossly both the lungs were congested and firm.

Histopathology report had a significant positive finding of presence of vegetable matter in the terminal bronchioles and alveoli, in sections studied from both lungs. These foci were surrounded by minimal leucocytes. As mentioned in Knight's forensic pathology, according to Gardner, even histological evidence of leucocyte clustering around foci of gastric contents deep in bronchi is an early postmortem event and not a vital reaction^[4]. But in present case considering the fact that the food material was embedded in mucous secretions along with expanded alveoli and broken septa, which is possible because of forced expiratory efforts and coughing as a result of obstruction in the respiratory passages, the leucocyte clustering around the vegetable foci can be considered as vital reaction of antemortem nature and the poor

inflammatory response, in an ante mortem aspiration, can be explained by occurrence of sudden death from choking leaving no time for inflammation to develop. **So the food material found in the respiratory tract was ante mortem phenomenon.**

2. Whether such autopsies should be labeled as negative autopsies or a questionable cause of death i.e. whether choking can be given as the cause of death?

As the vegetable matter embedded in mucous being present in the terminal bronchioles and alveoli along with the minimal leucocyte cells surrounding it substantiated the aspiration to be ante mortem and recent in the present case and as there was no any other positive finding on autopsy and viscera analysis, the cause of death can be attributed to choking.

3. If choking is the cause of death, what is the mechanism of death?

Asphyxia is the most common mechanism to cause death in choking, but there are cases where the immediate result of choking is vagal inhibition and sudden death. The diagnosis is difficult due to absence of overt signs^[6]. Several cases have been reported by Polson et al, wherein witnessed cases of sudden death showed a quantity of vegetable material, part of the lunch, obstructing the air passages downwards from the larynx to the intrapulmonary bronchi of both the lungs at postmortem examination. In all such cases, true signs of asphyxia were absent and death had been attributed to vagal inhibition^[8]. In the present case there were no signs specific for asphyxia and hence the mechanism of death due to choking can be attributed to vagal inhibition.

4. What is the reason of aspiration of gastric contents in the respiratory tract?

Choking can occur when vomited material is inhaled by a victim in a state of unconsciousness or under the influence of drink, drug, and anesthesia or during the fit of epilepsy^[9]. In the present case the deceased was not on medication, neither he was under the influence of alcohol, drug or anesthesia. Hence none of these factors can be attributed to present case. The patient was only suffering from gastro esophageal regurgitation as per history. A large, fatty meal, lying down, bending over or bending and lifting predisposes reflux^[10]. In the present case deceased might have vomited and subsequently aspirated the vomit. The exact triggering factor for gastric regurgitation and subsequent aspiration could not be ascertained and can only be hypothesized as there was no eyewitness to the terminal event. The deceased had gone to work place immediately after his lunch. The kind of work he was doing was plucking of grass weeds from the farming area, in which squatting, bending and lifting is involved. Probably from application of pressure onto his epigastric region due to bending and lifting immediately after his meals, led to regurgitation and subsequent aspiration of the stomach contents into his respiratory tract. The sudden entry of foreign material could have resulted into neurogenic cardiac arrest leading to his death.

Conclusion

An autopsy surgeon must be well-versed with phenomenon of artefacts to give conclusive opinion regarding cause of death. A dilemma regarding- gastric contents in respiratory tract whether artefact or cause of death can be solved by analyzing the autopsy findings, history, histopathological examination reports and chemical analysis findings, along with good amount of literature review.

References

1. Agarwal SS, Kumar L, Chavali KH. Legal Medicine Manual. 1st ed. New Delhi: Jaypee Brothers Medical Publishers Pvt. Ltd; 2008.
2. Agarwal SS, Kumar L, Malur PR, Singanagutti ST, Chavali KH. Gastric Contents In Respiratory Tract, A Diagnostic Dilemma At Autopsy. JIAFM 2010; 32(1):22-24.
3. Reddy KSN, Murty OP, The Essentials of Forensic Medicine. 32nd ed. Hyderabad: K SUGUNA DEVI; 2013.
4. Saukko P, Knight B. Knight'S Forensic Pathology. 3rd ed. London: Arnold; 2004.
5. DiMaio VJ, DiMaio D. Forensic Pathology. 2nd ed. Florida: CRC Press; 2001.
6. Diksit PC. Textbook of Forensic Medicine and Toxicology.2nd ed. New Delhi: PEPEE Publishers and distributers (P) Ltd; 2014
7. Mathiharan K, Patnaik AK. Modi's Medical Jurisprudence and Toxicology. 23rd ed. New Delhi: Lexis Nexis; 2005.
8. Polson CJ, Gee DJ. Essentials of Forensic Medicine. 3rd ed. Oxford: Pergamon Press; 1963.
9. J.B.Mukherjee's FORENSIC MEDICINE AND TOXICOLOGY.4thed.Kolkata: ACADEMIC PUBLISHERS; 2011.
10. Dr. Roger Henderson. Gastro esophageal reflux (Acid reflux). [Cited 2013 June 28] Available on: www.netdoctor.co.uk/diseases/facts/gastroesophagealreflux.htm.

Case Report

DEATHS DUE TO CHOKING AMONG PSYCHIATRIC PATIENTS A REPORT OF THREE CASES

Dr. CV Tingne, Dr. NB Kumar, Dr. PS Ghormade, Dr. RK Gadhari, Dr. AN Keoliya

Authors

Dr. Chaitanya Vidyadhar Tingne

Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr. Narendra Baluram Kumar

Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr. Pankaj Suresh Ghormade

Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr. Ramesh Kashinath Gadhari

Assistant Professor, Department of Forensic Medicine & Toxicology, SBH Government Medical College, Dhule, Maharashtra, India

Dr. Ajay Narmadaprasad Keoliya

Professor and Head, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Number of Pages: Four

Number of Tables: Nil

Number of Graph: Nil

Number of Photographs: Three

Corresponding Author:

Dr. Chaitanya V. Tingne
First floor, Plot no. 70,
West High Court Road, Bajaj Nagar,
Nagpur, Maharashtra, India, 440010
ctingne@gmail.com, 9822864460

Case Report

DEATHS DUE TO CHOKING AMONG PSYCHIATRIC PATIENTS A REPORT OF THREE CASES

Dr. CV Tingne, Dr. NB Kumar, Dr. PS Ghormade, Dr. RK Gadhari, Dr. AN Keoliya

Abstract

Choking is a form of asphyxia caused by an obstruction within the air passages most commonly by a food bolus lodging in the larynx or laryngopharynx. Choking incidents have long been recognized as a cause of death in people with mental illness and is one of the mechanisms postulated to connect the use of antipsychotic medication with sudden death. Choking deaths, if observed, are sudden and dramatic, and their common association with active eating is such that a report of the death is made to the authorities. We present here a report comprising of three psychiatric patients who died of accidental choking while eating.

Key words: Choking, psychiatric, atypical antipsychotics

Introduction

Choking is a form of asphyxia caused by an obstruction within the air passages most commonly by a food bolus lodging in the larynx or laryngopharynx. A large foreign body may be impacted in the pharynx and cause death from hypoxia. A small object partially blocking the lumen of the larynx may cause death by laryngeal spasm. Irritation of tracheal mucosa by foreign body may cause death by reflex parasympathetic cardiac inhibition. Patients with serious mental illness, especially those who are elderly, have poor dentition and eating habits, or take antipsychotics with anticholinergic and/or dopamine blocking effects, are at an increased risk of death due to choking. Choking incidents have long been recognized as a cause of death in people with mental illness and is one of the mechanisms postulated to connect the use of antipsychotic medication with sudden death^[1]. In addition to psychiatric patients; groups with higher choking risk include young children, the elderly, especially those with dementia, learning disabilities, intellectual disabilities and neurological patients. Some of them share the similar problem of imperfect mental status. In India the law mandates reporting to the authorities of any death that has occurred while mentally ill patients are held involuntarily in the care of a mental health service. Choking deaths, if observed, are sudden and dramatic, and their common association with active eating is such that a report of the death is made to the authorities. We present here a case report comprising of three psychiatric patients who died of accidental choking while eating.

Case report 1

A dead body of 68 year old female was brought for medicolegal postmortem examination. The deceased was mentally ill, undergoing treatment at Regional Mental Hospital. The total daily doses for the psychoactive medication regimen included Risperidone 2 mg daily for psychosis and mood lability and Clozapine 500 mg for mood lability. As per history narrated by the hospital staff, she had bouts of coughing and respiratory distress while she was taking food. She collapsed on the floor and in spite of prompt resuscitative measures could not be revived and declared dead.

Externally, the subject was averagely built; postmortem staining was present and fixed over the back and dependant parts of the body. There was bluish discoloration of finger and toe nails and lips. There were no external injuries seen. Internally all viscera were

congested. Stomach contained undigested food particles. Identical food bolus was present in larynx completely occupying the lumen (figure 1) with blood stained froth in trachea. Lungs



were oedematous exuding dark red color bloody fluid. Viscera were preserved and sent for chemical analysis of common poisons and any sedative drugs. Viscera were also sent for histopathological examination to rule out other pathologies. Both the analyses gave negative results. The cause of death was due to

Fig 1: Food bolus completely occluding the laryngeal opening asphyxia due to choking. Circumstances were consistent with accidental choking.

Case report 2



Fig 2: Piece of cottage cheese in infra glottic region

A 62 years old female corpse was brought for autopsy to the mortuary. She had a 22 year history of paranoid schizophrenia and was being treated with Olanzapine for many years. Two weeks ago, she had stopped taking her medication and was brought to a State run psychiatric hospital because of worsening paranoia and hostility. She was a known case of type II diabetes and poor dentition. She has no history of substance abuse. Just before her death, a few minutes after beginning her meal, she suddenly stood up and put her hands to her throat. She looked frightened, was not able to speak and became unconscious. She was then taken to the emergency department, where she was declared dead.

On external examination she was well built and nourished weighing 81 kg. There were no external injuries. Cyanosis was evident over fingernail beds and palms. Internally all organs were congested. A white colored partially chewed piece of cottage cheese was found occluding the laryngeal lumen (figure 2). Trachea contained semisolid food particles. Identical undigested food particles were present in stomach. Histopathological examination of organs and chemical analysis of viscera were non significant. The cause of death was respiratory and cardiac failure secondary to choking and foreign body obstruction.

Case report 3:

A corpse of 75 year old male was brought for medicolegal postmortem examination with an alleged history of sudden death while having breakfast. The deceased had a 40 year

history of paranoid schizophrenia, which has been well controlled with Olanzapine, 20 mg/d, for many years. He was a known case of hypertension and ischaemic heart disease and was undergoing treatment at Regional Mental Hospital since four decades. The deceased while eating breakfast abruptly stood up and put his hand against his throat and collapsed. In spite of resuscitative measures he died within an hour of the onset of symptoms.



Fig 3: Aspirated food material in the right main bronchus

At autopsy on external examination the deceased was a thin built male weighing 47 kg. Pieces of partially chewed up bread were present in the oral cavity. Internal examination revealed brown colored piece of bread occluding the lumen of larynx. Whitish semisolid food particles mixed with mucus were present in the trachea and right principal bronchus (figure 3). Stomach contained undigested food material identical to that found in trachea and larynx. Signs suggestive of asphyxia were present.

Routine viscera chemical analysis and histopathological examination of organs failed to reveal any associated pathology. The cause of death was asphyxia due to choking.

Discussion

Death from choking can be the result of pure hypoxia from occlusion of the airway, when all the attendant signs of congestion, cyanosis and perhaps petechiae may be present, usually where the victim struggles to breathe for an appreciable period. However a large proportion of deaths occur suddenly before any possible hypoxic manifestations have time to take effect; these fatalities must be caused by neurogenic cardiac arrest, either purely neurogenic or accelerated by excess catecholamine release from the adrenaline response^[2].

Reports on serious impairment of swallowing mechanism attributable to the side effects of psychotropic drugs appeared in literature soon after the introduction of phenothiazines^[3]. Psychiatric and geriatric patients often tend to have difficulty swallowing food particles, thus predisposing them to choking and aspiration^[4]. The high incidence of tachyphagia among patients with schizophrenia has been noted, as has the frequent abnormality of such patients' swallowing mechanisms. This could predispose individuals with schizophrenia to choking^[5].

All the three patients in the case report were elderly and were on atypical antipsychotics. Some authors have pointed out how the well-known depression of the bulbar centers, variously brought about by all kinds of psychotropic medication, causes, among other things, an inhibition of the cough reflex, the swallow reflex, and the gag reflex. Moreover, patients on psychotropic drugs may theoretically have a combination of:

- 1) dopaminergic blockade with peripheral and central effects on deglutition (block or timing upsets in the components involved in the swallowing process) and
- 2) cholinergic blockade with potential impairment of esophagus motility and the gag reflex.

Neuroleptics, antidepressants, and antiparkinsonians are often used together and have an anticholinergic synergetic effect on the swallowing process. However, no direct, clear-cut

connection has ever been shown between psychotropic drugs and fatal asphyxiation. Clinical experience, single case reports, epidemiological surveys, and pathophysiology studies, however, do suggest that they may play a key role through diverse pathogenetic mechanisms [6].

Psychotropic medications sometimes increase appetite or food craving through an effect on hypothalamus, through leptin and ghrelin metabolism. Some elderly patients, chronic schizophrenic and mentally retarded patients had poor dental health that might further impair the quality of food boluses to be swallowed [7].

Patients on psychotropic medications should be reviewed regularly for side-effects, notably extrapyramidal and anticholinergic side-effects. Patients should be advised to eat slowly if they have a problem with overeating and tachyphagia. Relatives should be alerted to the possible choking risk in high-risk patients, and advised to feed those in their care slowly with appropriate types and amounts of food. Nursing staff should be trained to identify patients at high risk of choking and provide appropriate resuscitation measures if such incidents occur. Analyses of choking incidents and regular audit can help to monitor trends and implement effective risk management measures.

References

1. Hollister LE. Unexpected asphyxial deaths and tranquilizing drugs. *AJ Psychiatry*. 1957;114,1035-1038.
2. Burns A, Horan M, Clague J, McLean G. *Geriatric Medicine for Old Age Psychiatrists*. Taylor and Francis, London New York 2006; 193-196.
3. Von Brauchitsch H, May W. Deaths from aspiration and asphyxia in a mental hospital. *Arch of Gen Psych*. 1968; 18,129-136.
4. Simpson GM, Davis JM, Jefferson JW. Sudden Death in Psychiatric Patients: The Role of Neuroleptic Drugs. American Psychiatric Association Task Force Report; 1987 27. Washington,DC: APA.
5. Saukko P, Knight B. Suffocation and asphyxia. In Saukko P, Knight B eds. *Knight's Forensic Pathology*. 3rd ed. London. Hodder Arnold. 2004;pp- 352-365.
6. Fioritti A, Giaccotto L, Melega V. Choking Incidents among Psychiatric Patients: Retrospective Analysis of Thirty-one Cases from the West Bologna Psychiatric Wards. *Can J Psychiatry*, June 1997, 515-520.
7. PHW Yim, CSY Chong. Choking in Psychiatric Patients: Associations and Outcomes. *Hong Kong J Psychiatry* 2009;19:145-149.

Case Report

DELAYED DEATH DUE TO FATAL PULMONARY EDEMA IN NEAR HANGING

Dr. NB Kumar, Dr. CV Tingne, Dr. PS Ghormade, Dr. AN Keoliya

Authors

Dr. Narendra Baluram Kumar, Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr. Chaitanya Vidyadhar Tingne, Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr. Pankaj Suresh Ghormade, Assistant Professor, Department of Forensic Medicine & Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Dr. Ajay Narmadaprasad Keoliya, Professor and Head, Department of Forensic Medicine and Toxicology, Indira Gandhi Government Medical College, Nagpur, Maharashtra, India (440018)

Number of Pages: Three

Number of Tables: Nil

Number of Graph: Nil

Number of Photographs: Three

Corresponding Author: Dr. Narendra Baluram Kumar
513. Bhrammasiddhi Building, S.H. Tandel Marg
Century Bazaar, Worli, Mumbai, 400025
naren2014@gmail.com, 9890876273

Case Report

DELAYED DEATH DUE TO FATAL PULMONARY EDEMA IN NEAR HANGING

Dr. NB Kumar, Dr. CV Tingne, Dr. PS Ghormade, Dr. AN Keoliya

Abstract

Hanging is the commonest mode of suicide in India. It is a painless method of committing suicide and death is instantaneous. Only few persons survive this episode, if rescued promptly and usually die at a later stage, which more precisely can be called delayed hanging death. Delayed death in hanging being a rare phenomenon has been reported infrequently in the literature. We report a case of a young adult male who was rescued within minutes of hanging but succumbed to the consequent complications.

Keywords: Near hanging, delayed death, suicide, pulmonary edema,

Introduction

Hanging has become the commonest method of suicide in India followed by poison ingestion. Statistics show an increasing incidence in suicidal hanging over past three years. In the year 2012, 37.0 % people committed suicide by hanging which is 4 % more than the previous year (33.2%).¹ Hanging is defined as death due to external pressure on the neck when a ligature is applied to the neck of a wholly or partly suspended individual. The term "near hanging" refers to patients who survive a hanging injury long enough to reach the hospital.^{2,3}

There are a number of mechanisms by which hanging may cause death, which may act independently or in concert. These include: stretching of the carotid sinus causing reflex cardiac arrest; occlusion of the carotid (and possibly vertebral) arteries; venous occlusion; airway obstruction resulting from pushing the base of the tongue against the roof of the pharynx or from crushing of the larynx or trachea; and finally spinal cord- brainstem disruption.⁴ In hanging delayed death occurs due to aspiration pneumonia, infection, hypoxic encephalopathy, oedema of the lungs, oedema of larynx, infarction or abscess of the brain and cerebral softening.⁵ We present a case of a young adult male who developed fatal complications even though he was rescued within minutes of attempted hanging.

Case report

A 27 year old male was brought to emergency department with altered sensorium, restlessness and in gasping state with an alleged history of attempted suicidal hanging. He was rescued by his neighbors within minutes of hanging and was brought to hospital within 20 minutes. He was suffering from depression since 2 years and was under medication for the same. He was admitted in intensive care unit and put on a ventilator. He was aggressively managed and even though his sensorium improved to some extent he succumbed to the complications on 6th day. A medicolegal autopsy was performed on the deceased at the Department of Forensic Medicine, Indira Gandhi Government Medical College, Nagpur.

On external examination an obliquely placed ligature mark was present over the neck, above the level of thyroid cartilage running backwards and upwards towards nape of neck having dimensions 23 cm x 2 cm. It was a healing abrasion, with a brownish dried scab surrounded by a puckered skin. Mark was non-continuous for 8 cm over the back of neck. It was situated 5.5 cm below chin, 4 cm below tip of right mastoid and 3cm below tip of left mastoid process. Therapeutic intravenous injection marks were present over dorsum of both hands.

On internal examination all organs were congested. Brain was soft and edematous. Lungs were edematous weighing 550gms (right) and 480gms (left); on cut section oozing of fine frothy fluid was present along with red to grey hepatization in lower lobes of both lungs. Stomach was empty without any peculiar odour. All neck structures were intact. Histopathological samples from lungs revealed edema (Photo 1) and evidence of pneumonitis (Photo 2) while those from brain had evidence of edema (Photo 3). No specific pathology was observed in heart and kidneys.

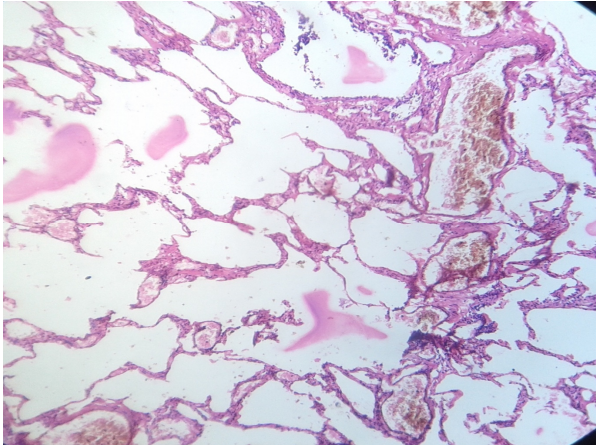


Photo 1: Lung showing congestion and oedema

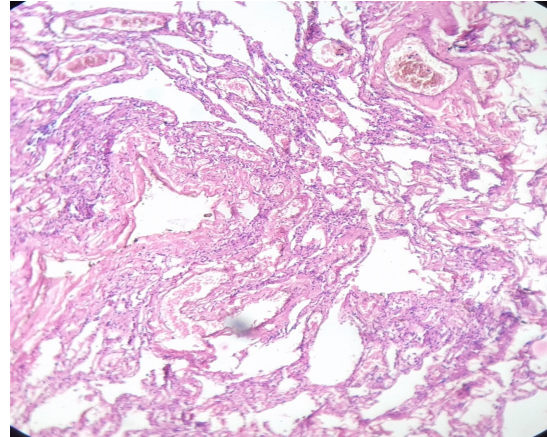


Photo 2: Lung showing congestion and interstitial mononuclear infiltration

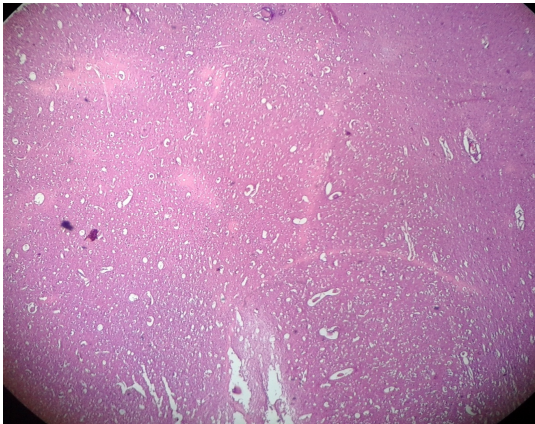


Photo 3: Brain showing congestion and oedema

Discussion

Victims of hanging usually die within a period of three to five minutes. Death in hanging occurs earliest when there is dislocation of the cervical vertebrae and injury to medulla leading to failure of respiratory centre. Deaths due to closure of cervical and vertebral arteries too occur quickly but not as quick as in injury to cervical vertebrae. Ligature at the level of cricoid cartilage causes complete asphyxiation earliest. When the level is over larynx the fatal period is slightly more and is maximum when the ligature is between chin and hyoid.⁶

In suicidal hangings, there is often no or minimal drop height. Compression of the soft tissues of the neck results in jugular venous obstruction (stagnant hypoxia) and loss of consciousness.⁷ The body then becomes limp from loss of muscle tone, which further tightens the ligature around the neck, resulting in carotid arterial obstruction, with or without airway closure, cerebral hypoxia and death.⁸ The duration of hanging correlates with outcome, and several small studies have shown hanging times of less than 5 minutes predicts good outcome.^{9,10}

Delayed deaths in hanging ranging from 18 hours to 39 days have been described in literature. In the present case the deceased was rescued within minutes of hanging and was brought to a tertiary care centre within 20 minutes. He had altered sensorium and respiratory distress on admission. He was resuscitated and aggressively managed in ICU. Slight

improvement was seen in sensorium but respiratory distress owing to pulmonary edema and pneumonitis could not be countered to the extent of successful revival and he succumbed on 6th day of admission.

Most of the patients develop respiratory and neurological complications immediately after the incident, the severity of which varies proportionally to the quickness of ligature removal. Pulmonary oedema as observed in our case is one of the most common immediate complication in patients following their rescue from acute airway obstruction or suicidal hanging.¹¹⁻¹³ Its onset is very rapid, generally appears within minutes of the event but sometimes it may be delayed. The cause of delay is not clear but it might be related to rate of onset of oedema and severity of airway obstruction.¹³ The exact mechanism of development of pulmonary oedema after rescue from hanging or strangulation is still not clear.

Conclusion

Delayed hanging death occurs due to a variety of mechanisms. Pulmonary edema is one of them. Survival of such victims not only depends on the early relieving of neck compression but also on prompt resuscitation measures. This case emphasizes the importance of forensic medicine experts to give final opinion regarding the cause of death by post mortem examination in cases of delayed hanging.

References

1. Crime in India. National Crime Records Bureau. Ministry of Home Affairs. [Internet] 2014 June 15. Retrieved from: <http://ncrb.nic.in>.
2. Adams N. Near hanging. *Emerg Med*. 1999;11:17–21.
3. McHugh TP, Stout M. Near-hanging injury. *Ann Emerg Med*. 1983;12:774–6.
4. Saukko P, Knight B. Suffocation and asphyxia. In Saukko P, Knight B eds. *Knight's Forensic Pathology*. 3rd ed. London. Hodder Arnold. 2004;pp-352-365.
5. Reddy KSN. Mechanical asphyxia. In *The Essentials of Forensic Medicine and Toxicology*. 32nd ed. Hyderabad. Om Sai. 2010;pp-323
6. Nandy A. Violent asphyxial deaths. In *Nandy's Handbook of Forensic Medicine and Toxicology*. 1st ed. New Central Book Agency (P) Ltd. 2013: pp 327-355.
7. Pesola GR, Westfal RE. Hanging-induced status epilepticus. *Am J Emerg Med* 1999;17:38-40.
8. Iverson KV. Strangulation: a review of ligature, manual, and postural neck compression injuries. *Ann Emerg Med* 1984;13:179—85.
9. Matsuyama T, Okuchi K, Seki T, Murao Y. Prognostic factors in hanging injuries. *Am J Emerg Med* 2004;22:207—10.
10. Vander Krol L, Wolfe R. The emergency department management of near-hanging victims. *J Emerg Med* 1994;12:285—92.
11. Oswalt CE, Gates GA, Holmstrom Pulmonary edema as a complication of acute air way obstruction. *JAMA*. 1977;238:1833–35.
12. Mantha S, Rao SM. Noncardiogenic pulmonary oedema after attempted suicide by hanging. *Anaesth*. 1990;45:993– 94.
13. Lang SA, Duncan PG, Shephard DA, Ha HC. Pulmonary oedema associated with airway obstruction. A Review. *Can J Anaesth*. 1990;37:210–18.

Case Report

PYRETHROID POISONING: AN UNCOMMON DEATH

Dr. R. Ravi Kumar, Dr. Kalai Selvi

Author:

Dr. R. Ravi Kumar, M.D, Assistant Professor, Department of Forensic Medicine, Rajarajeshwari Medical College and Hospital, Kambipura Mysore road, Bangalore – 74, dr_ravikumar_fm@yahoo.com, Ph: 94483 87665

Dr. Kalai Selvi, L.T, Post Graduate Student, Department of Forensic Medicine. Rajarajeshwari Medical College Hospital, Kambipura, Mysore road, Bangalore-74

Number of Pages: Four

Number of Tables: Nil

Number of Photographs: Nil

Address for correspondence: Dr. R. Ravi kumar, M.D, Assistant Professor,
Department of Forensic Medicine,
Rajarajeshwari Medical College and Hospital,
Kambipura Mysore road, Bangalore – 74,
dr_ravikumar_fm@yahoo.com, Ph: 94483 87665

Case Report

PYRETHROID POISONING: AN UNCOMMON DEATH

Dr. R. Ravi kumar, Dr. Kalai Selvi

Abstract

In developing countries like India most common method adapted for committing suicide is poisoning, especially in rural areas where the availability of poisons are quite easy in the form of insecticides or fertilizers. Pyrethroid is one such biological insecticide which is most widely used as commercial and household insecticide. Even though it is rare it can be used to commit suicide. Features of atypical presentations can occur due to the unauthorized pyrethroid/organophosphate mixtures marketed in some developing countries. Death by pyrethroid poisoning is not very common alike organophosphorus compounds, but still it is documented in rural parts of our country. The signs and symptoms simulate the organophosphorous compound poisoning and there is a chance of misdiagnosis. Here we are presenting a fatal case of suicidal pyrethroid poisoning, which is relatively rare but it will pose problems in diagnosis and management.

Keywords: Suicide, pyrethroid poisoning, pyrethrin poisoning, biological insecticide

Introduction

In developing countries, acute poisoning from pesticides is the most worrisome type of poisoning. However in developed countries acute pesticide poisoning is controlled.^[1] Pyrethrum is a naturally occurring mixture of chemicals found in chrysanthemum flowers. Active insecticidal properties in the pyrethrum extract compounds are called pyrethrins. Pyrethroids are manufactured chemicals that are very similar in structure to the pyrethrins, but are often more toxic to insects, as well as to mammals, and last longer in the environment than pyrethrins. Pyrethrins and pyrethroids are often combined commercially with other chemicals called synergists, which enhance the insecticidal activity of the pyrethrins and pyrethroids. The synergists prevent some enzymes from breaking down the pyrethrins and pyrethroids, thus increasing their toxicity.^[2] Although many studies have shown that pyrethroids are less toxic to humans compared to other insecticides, we cannot define or determine the outcome accurately.

Case Report

As per the investigating officer's report the deceased, a male aged 30 years, had consumed mosquito repellent insecticide intentionally at his residence and he was brought to the hospital to the emergency department and was admitted. As per hospital records he presented with nausea, abdominal pain and 5 episodes of vomiting. Vitals were stable. Stomach wash was given, Intravenous injection of atropine was started with other supportive treatment. All the routine investigations including cholinesterase levels were normal. After 3 days the deceased was drowsy and disoriented following which he developed breathlessness, hypotension and was intubated and put on mechanical ventilator. In spite of these measures the patient succumbed to death. Cause of death was cardio-respiratory failure due to alleged insecticide poisoning. The next day postmortem was conducted. External examination was unremarkable. On internal examination, stomach contained 50 ml of red colour fluid. Mucosa was congested. Both lungs were oedematous and showed features of consolidation. Liver showed fatty changes. All other internal organs were intact and congested. Blood, stomach and contents and part of liver and kidneys were sent for chemical analysis. Chemical analysis

report confirmed the presence of pyrethroid insecticide. Cause of death is due to respiratory failure as a result of consumption of substance containing pyrethroid insecticide.

Discussion & Review of Literature

Suicide is the act of intentionally causing one's own death, that is the "act of taking one's own life"^[3] Around 800,000 to a million people die by suicide every year, making it the 10th leading cause of death worldwide^[4,5] Common methods include: hanging, pesticide poisoning, and firearms.

Self-poisoning with agricultural pesticides represents a major hidden public health problem accounting for approximately one-third of all suicides worldwide.^[6] It is one of the most common forms of self-injury in the Global South. Most cases of intentional pesticide poisoning usually due to sudden impulsive thoughts/acts during stressful events, and not only that even the availability of pesticides strongly influences the incidence of self poisoning.

Pyrethroids are widely used as commercial and household insecticides.^[7] Among the pyrethroids are the synthetic pyrethroids, is one such newest biological pesticide to enter the marketplace. In the concentrations used in such products, they also have insect repellent properties and are generally harmless to human beings in low doses but can harm sensitive individuals.^[8] Bifenthrin, Fluvalinate, Cyfluthrin, Permethrin, Esfenvalerate, Phenothrin are few chemical names of Pyrethroid Insecticides.^[9]

Mechanism of action of Pyrethroids are axonic excitoxins the toxic effects of which are mediated by preventing the closure of the voltage-gated sodium channels in the axonal membranes. When the toxin keeps the channels in their open state, the nerves cannot repolarize, leaving the axonal membrane permanently depolarized, thereby paralyzing the organism.^[10] Fatal dose is 1gm/ kg body weight.^[11]

Signs of pyrethroid poisoning: although pyrethroids have been used for many years, there have been few reports of systemic poisoning by these compounds. This is because, although they are absorbed as other pesticides, they are quickly broken down to harmless products in the body after absorption.^[12]

Type II acute poisonings are generally more severe than Type I.^[13] Type I poisoning has been described as characterized by fine tremor and reflex hyperexcitability. Type II poisoning has typically shown severe salivation, hyperexcitability and choreoathetosis. Other signs and symptoms of toxicity include abnormal facial sensation, dizziness, headache, fatigue, vomiting, diarrhea and irritability to sound and touch. In more severe cases, pulmonary edema, muscle fasciculations, seizures and coma can develop.^[14] Pyrethroids are not cholinesterase inhibitor. However there have been some cases in which pyrethroid poisoning is misdiagnosed as organophosphorus poisoning due to similar presenting signs.^[15] Some commercial products also contain organophosphorous or carbamate insecticides in that time person can have mixed signs. common cause of death in cases of pyrethroid poisoning are allergic reactions, respiratory failure (hypersensitivity pneumonitis, pulmonary oedema), seizuers, secondary pneumonia, coma.

Atypical presentations can occur with the pyrethroid poisoning where patients present with respiratory failure requiring mechanical ventilation, hypotension, pneumonia, acute kidney injury and seizure.

Treatment includes skin decontamination, airway protection, gastrointestinal decontamination and seizure treatment.^[16] As there is no specific antidote, early diagnosis and aggressive supportive therapies are the only remedies to prevent mortality.

In our patient a clear history of mosquito repellent ingestion was available. Physicians working in emergency departments or ICUs should be aware of this particular poisoning

which can clinically mimic OP poisoning, however, to differentiate these two kinds of pesticide poisoning, exposure history is more important.

In general, pyrethroid has been known to be relatively benign in mammals, however atypical presentations have occurred in poisoned patients.^[17] Most common atypical presentation in this case study was signs and symptoms associated with gastrointestinal tract followed by respiratory failure requiring ventilator care. We thought respiratory failure was secondary to respiratory depression due to acute lung injury.

Poisoning due to pyrethroids clinically resemble poisoning due to common insecticides like organophosphates and this can lead to misdiagnosis. Moreover, there is no inhibition of plasma cholinesterase in pyrethroid poisoning and requirement of atropine is usually less than 10mg. It is very essential to differentiate between the two, as few cases of death have been reported due to atropine toxicity.^[14] Excess atropine causes agitation, confusion, urinary retention, hyperthermia and tachycardia.

Conclusion

Diagnosis of pyrethroid poisoning is mainly based on exposure history, circumstantial evidence, clinical notes in corroboration with findings at autopsy.^[18] The common atypical presentation was respiratory failure requiring ventilator care. Most treating physicians can misdiagnose pyrethroid poisoning as organophosphorus poisoning due to the fact that they are easily available and used among farmers and agricultural workers and the other reason is the signs and symptoms of the pyrethroid poisoning simulate the organophosphorus poisoning. In conclusion, mosquito repellent insecticide poisoning though rarely reported can be much frequent in occurrence due to its easy accessibility in households.

Acknowledgement

Work attributed to Department of Forensic Medicine at the Rajarajeshwari medical college hospital, Bangalore, Karnataka Their assistance is gratefully acknowledged.

References

1. http://www.ipm-info.org/library/documents/jeryaratnam_who1990_acute_poisoning.pdf
2. G. Daniel Todd, David Wohlers, Toxicological Profile For Pyrethrins And Pyrethroids, U.S. Department Of Health And Human Services Public Health Service Agency For Toxic Substances And Disease Registry, September 2003
3. Stedman's medical dictionary (28th ed. ed.). Philadelphia: Lippincott Williams & Wilkins. 2006. ISBN 978-0-7817-3390-8. <http://en.wikipedia.org/wiki/Suicide>
4. Hawton K, van Heeringen K (April 2009). "Suicide". *Lancet* 373 (9672): 1372–81. doi:10.1016/S0140-6736(09)60372-X. PMID 19376453 <http://en.wikipedia.org/wiki/Suicide>
5. Värnik, P (2012 Mar). "Suicide in the world.". *International journal of environmental research and public health* 9 (3): 760–71. doi:10.3390/ijerph9030760. PMC 3367275. PMID 22690161.
6. <http://bjp.rcpsych.org/content/189/3/201.short>
7. Robert L. Metcalf "Insect Control" in Ullmann's Encyclopedia of Industrial Chemistry" Wiley-VCH, Weinheim, 2002. doi:10.1002/14356007.a14_263 <http://en.wikipedia.org/wiki/Suicide>
8. Pyrethroids fact sheet from the Illinois Department of Public Health. <http://en.wikipedia.org/wiki/Suicide>
9. WPS FACT SHEET 4/01. New Jersey Department of Environmental Protection Pesticide Control Program. PO Box 420 Trenton, NJ 08625-0420

10. D. M. Soderlund, et al., "Mechanisms of pyrethroid neurotoxicity: implications for cumulative risk assessment", *Toxicology* 2002 171, 3-59. doi:10.1016/s0300-483x(01)00569-8
11. Reddy KSN. *The Essentials of Forensic Medicine and Toxicology*. 31st Edition. published by K. Suguna Devi. 2012; Page-491.
12. Network for sustainable agriculture. agLe@rn.net
Ray DE, Foreshaw PJ. Pyrethroid insecticides: poisoning syndromes, synergies and therapy. *J Toxicol Clin Toxicol*. 2000;38(2):95-101
13. He F, Wang S, Liu L, Chen S. Clinical manifestations and diagnosis of acute pyrethroid poisoning. *Arch Toxicol*. 1989;63(1): 54-58.
14. Ray DE, Fry JR. Reassessment of the neurotoxicity of pyrethroid insecticides. *Pharmacol Ther*. Jul 2006;111(1):174-193
15. <http://www.biomedcentral.com/1471-2458/7/357/> Reigart, J.R. and Roberts, J.R. (1999). *Recognition and Management of Pesticide Poisonings*. Washington, DC: Environmental Protection Agency.
16. Yang PY, Lin JL, Hall AH, et al. Acute ingestion poisoning with insecticide formulations containing the pyrethroid permethrin, xylene and surfactant: a review of 48 cases. *J Toxicol Clin Toxicol*. 2002;40:07-13.
17. Dr. VP Kumar Sriperumbuduru, Dr. Seetharamaiah Mynedi. Pyrethroid Poisoning In Clinical And Medico-legal Perspective - An Overview And Case Report. *Int J Biol Med Res*. 2013; 4(3):3493-3494 Journal homepage: www.biomedscidirect.com

Case Report

A CONFLICTING CASE OF STRANGULATION: A CASE REPORT

Dr. SD Wakde, Dr. KU Zine, Dr. PG Dixit

Authors

Dr. S. D. Wakde, Assistant Professor, Department of Forensic Medicine, Government Medical College and Hospital, Nagpur, Maharashtra

Dr. P. G. Dixit, Professor and Head, Department of Forensic Medicine, Government Medical College and Hospital, Nagpur, Maharashtra.

Dr. K. U. Zine, Professor and Head, Department of Forensic Medicine, Government Medical College and Hospital, Aurangabad, Maharashtra.

Number of Pages: Three

Number of Tables: Nil

Number of Graph: Nil

Number of Photographs: Four

Corresponding Author: Dr. Shailesh D. Wakde,
Plot No.43/A, New Prerna Nagar,
New Narsala Road, Near Mahalgi Nagar,
Mahalgi Nagar Post Office. Nagpur.
Pin: 440034 Maharashtra, India.
9552888199, sdwakde@gmail.com

Case Report

A CONFLICTING CASE OF STRANGULATION: A CASE REPORT

Dr. SD Wakde, Dr. KU Zine, Dr. PG Dixit

Abstract

A female aged 25 year was brought with a history of found unconscious at home, the body was sent to Government Medical College and Hospital, Aurangabad, Maharashtra for a medico legal post mortem. Deceased had a ligature mark over anterior aspect of neck suggesting it as a case of hanging accompanied by few external injuries like abrasions over face and chest consistent with fall at ground level. On conclusion of autopsy, the cause of death was found to be strangulation.

The process of strangulation, whether by hand (manual) or by ligature, results in blunt force injury of the tissues of neck. The pattern of these injuries allow us to recognize strangulation as a mechanism, and to distinguish strangulation from other blunt injuries including hanging, traumatic blows to the neck, and artifacts of decomposition.^[1-7]

These classical findings were absent in this case. Absence of these characteristic findings could easily misinterpret such a case as that of hanging. Therefore this case is presented.

Key words: Ligature mark, abrasions, neck tissue injuries, homicide.

Introduction

With its relatively small diameter, lack of bony shielding, and close association of the airway, spinal cord, and major vessels, the human neck is uniquely vulnerable to life-threatening injuries. Throughout recorded history, various methods of strangulation (i.e. disruption of normal blood and air passage in the neck) have been used by both assailants and penal systems to produce injury and death.^[8]

“Strangulation is the condition of violent asphyxia death in which, the exchange of air between the atmosphere and the lungs is prevented by way of constriction of neck by means of a ligature material or by some other means, without suspending the body of the victim, where the force of constriction is applied from outside (exogenous in origin) and is not the weight of body or the head of the victim.”^[9]

Case

On 17th October 2012, female aged 25 year with a history of found unconscious at home, was brought dead to Government Medical College and Hospital, Aurangabad Maharashtra. She was subjected to strangulation by use of sari, the body was sent for a medico legal post mortem.

Post Mortem Finding

The body was of normal built female. There were following injuries over the body.^[10]



Photo 1: Ligature mark over anterior neck.



Photo 2: Abraded contusions over inner lips

Ligature mark at front of the neck above the level of thyroid cartilage which was running horizontally backwards and upwards. Abrasion of size 0.4 x 0.5 cm at the tip of nose. Multiple abraded contusions ranging from 0.2 x 0.2 to 1 x 1.6 cm over inner aspect of lips. A small linear abrasion of length 1.cm over right para sternal region of chest, near xiphysternum. Abrasion over right mid submandibular region of size 1x0.6 cm. Contusion observed over upper one third of esophagus, over an area of 3x3 cm, which was the only significant internal autopsy finding going in favor of strangulation.



Photo 3: Neck dissection show clear field.

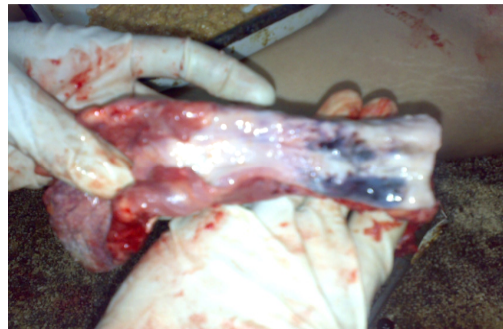


Photo 4: Contusion over upper esophagus

At the end of autopsy the cause of death was attributed to asphyxia due to ligature strangulation.

Discussion

Strangulation is differentiated from hanging by findings of superficial subcutaneous neck tissue injuries. The ligature mark is at or below the level of the thyroid cartilage. It is circular, continuous, abraded and contused and sometimes parchmentized. The mark of knot is usually in front but may be at any place. In many cases, ligature material is left around the neck after death of the victim. The face is highly congested and cyanosed. The eye may be partly open and the eyeballs and the tongue are protruded. Tardieu's spots are more abundant than in case of hanging and are present on the forehead, temples, eyelids, and under the conjunctiva. There may be wide areas of sub conjunctival hemorrhage. There may be bleeding from the nose. Involuntary discharge of urine and fecal matter is more common in cases of strangulation than hanging. Strangulation, being mostly homicidal, in most cases there may be presence of marks of resistance on the body. The most important internal findings lie in the neck. The subcutaneous tissue underneath the ligature mark is contused, often torn at a few places with gross extravasations. In case of strangulation by ligature, fracture of the superior horn of thyroid cartilage or subluxation between the two sides may be expected.^[9]

These classical findings were absent in this case. Absence of these characteristic findings could easily misinterpret such a case as that of hanging.

Conclusion

Deaths due to compression of neck are one of the most important areas of investigation of unnatural deaths encountered in day to day medico legal works.

The nature of violence at neck is so wide and varied that is challenging task for autopsy surgeon on many occasions. Therefore careful and meticulous study of every case is mandatory to bring out comprehensive / wide verities of observations in deaths due to compression of neck and also to differentiate the manner of deaths.

References

1. Kelly M: Trauma to the neck and larynx [Review]. *Crna* 8(1):22-30, 1997 Feb.
2. Missliwetz J, Vycudilik W: Homicide by strangling or dumping with postmortem injuries after heroin poisoning? *American Journal of Forensic Medicine & Pathology* 18(2):211-4, 1997 Jun.
3. Denic N, Huyer DW, Sinal SH, Lantz PE, Smith CR, Silver MM: Cockroach: the omnivorous scavenger. Potential misinterpretation of postmortem injuries. *American Journal of Forensic Medicine & Pathology* 18(2):177-80, 1997 Jun.
4. Samarasekera A, Cooke C: The pathology of hanging deaths in Western Australia. *Pathology* 28(4):334-8, 1996 Nov.
5. Ortman C, Fechner G: Unusual findings in death by hanging--reconstruction of capacity for action. [German] *Archiv fur Kriminologie* 197(3-4):104-10, 1996 Mar-Apr.
6. Howell MA, Guly HR: Near hanging presenting to an accident and emergency department. *Journal of Accident & Emergency Medicine* 13(2):135-6, 1996 Mar.
7. Maxeiner H: "Hidden" laryngeal injuries in homicidal strangulation: How to detect and interpret these findings. *J Forensic Sci* 43 (No. 4): 784-791, 1998 July.
8. William Ernoehazy Jr. Hanging Injuries and Strangulation Medscape: Online Article. Accessed on 08/10/2013. Available from: <http://emedicine.medscape.com/article/826704-overview>
9. Nandy A. Violent Asphyxial deaths. *Principles of Forensic Medicine including Toxicology*. New Central Book Agency (P) Ltd. 3rd Edi. (2010) 529-30.
10. P. M. No. 2029/12 dated. 17/10/2012.

Case Report

MORBID AUTOPSY FINDINGS DO REVEAL MOTIVE OF THE CRIME A CASE REPORT

Dr. D Ingale, Dr A Kanaki, Dr M Sane, Dr. A Mugadlimath,
Dr N Gupta, Dr. B Chandrashekhar

Authors

Dr. Dharmaraya Ingale, Professor & HOD, Dept of FMT, Shri B M Patil Medical College, Bijapur

Dr Ashok Kanaki, Associate Professor, Dept of FMT, Ashwini rural Medical College, Solapur

Dr Mandar Sane, Assistant Professor, Dept of FMT, Govt Medical College, Aurangabad

Dr. Anand Mugadlimath, Associate Professor, Dept of FMT, Ashwini rural Medical College, Solapur

Dr Neeraj Gupta, PG Student, Dept of FMT, Shri B M Patil Medical College, Bijapur

Dr. Bhuyyar Chandrashekhar, PG Student, Dept of FMT, Shri B M Patil Medical College, Bijapur

Number of Pages: Three

Number of Tables: Nil

Number of Graph: Nil

Number of Photographs: Nil

Corresponding Author: Dr. Dharmaraya Ingale, Professor & HOD,
Dept of FMT, Shri B M Patil Medical College,
Bijapur

Case Report

MORBID AUTOPSY FINDINGS DO REVEAL MOTIVE OF THE CRIME A CASE REPORT

Dr. D Ingale, Dr A Kanaki, Dr M Sane, Dr. A Mugadlimath,
Dr N Gupta, Dr. B Chandrashekhar

Abstract

A 45 year old widow was brought to community health centre with h/o sudden onset of chest pain, breathlessness at midnight from nearby village. The doctor on duty referred the case to District headquarters for further treatment after first aid. The case was later admitted and treated for alleged organophosphorus poisoning for 4 days in a private hospital. She was declared dead on 5th day early morning. Medico Legal post mortem examination was held at the request of Police, on the same day at noon. This case is reported for its unique findings contradicting the history furnished by the police and the relatives, revealing motive of crime.

Key words: Motive of the crime, Organophosphorus, decidual reaction

Introduction

Objectives of the medico legal autopsy are, to assist the investigating agencies in establishment of identity of the unknown deceased, to find exact cause of death, time since death, mode & manner of death and in case of newborns to decide live birth or dead birth⁽¹⁾. Often in routine practice of the medico legal autopsy, concern is to opine as to the exact cause of death & time since death. However the doctor should not volunteer to opine as to the manner of death on autopsy findings alone. Also, it should be remembered that detection of the motive of the crime is not within the purview of the doctor. Nevertheless, careful & complete medico legal autopsy and recording of all the wounds, diseases, anomalies, abnormalities, condition or the state of the organs do help in deciding motive of the crime.

Criminal abortion comprises the deliberate ending of pregnancy outside legal provisions. Hippocratic Oath banned the involvement of a doctor in abortion. In 1970, Oslo declaration modified Geneva declaration as allowing the doctor in abortion only when this was sanctioned by the laws of their native country⁽²⁾. Here is a case report of 45 year old widow, who had sudden onset of breathlessness at mid night and treated for suspected poisoning by organo-phosphorous compound for 4 days, declared dead at private hospital. The case was registered under 174 Cr PC and the dead body was subjected for medico legal post mortem examination, which revealed the facts.

Case report

The dead body of 45 year old widow was subjected for medico legal post mortem examination under Section 174 CrPC. As per the history furnished by the police in the requisition for the autopsy, the deceased had sudden onset of breathlessness & chest pain, she was rushed to government hospital nearby, from where she was referred to District headquarters for further treatment. As per the summary of treatment given by the hospital, she died 4 days after being treated for suspected organophosphorus poisoning. On enquiring, the relatives provided the information that there is family history of malignancy of uterus.

External examination

Moderately built & nourished female body, rigor mortis established all over, post mortem lividity present on the back of the trunk, nail beds were bluish. Intravenous injection

marks present over the back of the hands, Central venous catheterization present over the neck. There was froth around mouth & nostrils. There were multiple hemorrhages under the skin all over the body even amidst the post mortem staining. Eyes were congested, ischemic necrosis & blebs filled with serous fluid were present over the distal phalanx middle finger of left hand.

Internal examination

Brain was congested & edematous. Both the lungs were congested, edematous & consolidated patchily. Heart was congested & edematous, large vessels contained dark fluid blood. Stomach had 25 ml of brown color fluid without any abnormal smell, mucosa was congested & hemorrhagic. Liver showed macro nodular changes over its surface, spleen was enlarged and covered by white exudative material. Both the kidneys were congested. The peritoneal cavity contained two liters of straw color fluid. All the internal organs showed hemorrhagic areas on their surface and substance with hemorrhagic areas in the serosal layers & mesentery.

The uterus was congested, enlarged measuring 13x8x5cms containing blood and blood clots, the endometrium was thickened & hemorrhages were present over bilateral fundal and infundibular areas. Cervix was edematous, hemorrhagic & external os showed irregular inflamed areas. Both ovaries had serous cysts of 4 to 5 cms size on both side, which collapsed on cutting.

Chemical analysis of viscera revealed organophosphorus insecticide compound.

Histopathology revealed edema in lungs. Endometrium showed proliferative endometrial glands with deciduas reaction. Both the ovaries showed changes suggestive of enlarged follicles. Ovaries revealed presence of enlarged follicles

Discussion

Incompetent abortionists lack in hygiene, skills, aseptic procedures, literacy and safety. About 8000 deaths occur every year due to septic abortion⁽³⁾.

Suicide amongst females may be due to various reasons including family problems, love affairs, illness, illicit relation etc⁽⁴⁾. However, unusual reasons like illegitimate pregnancy may also creep up, as in present case. Finding a motive for suicide may not be prime objective of an autopsy, but ascertaining a reason behind suicide should be attempted for a greater social cause. In the present case, decidual reaction was noted in uterus and enlarged follicles in ovaries were noted. Decidual reaction is completed only with blastocyst implantation, which occurs not earlier than 6-7 days after fertilization⁽⁵⁾.

Looking at the morbid autopsy findings in the uterus and ovaries, it is certain that the widow woman had aborted in recent past, preceding the last illness. Now the question arises as to whether the abortion was induced or spontaneous. In absence of injuries to cervix, the autopsy findings are not specific to decide manner of expulsion of products of conception. However her last illness could be sequel to these findings in the form of disseminated intravascular coagulation as a complication of abortion. Alternatively, it could be of consequent to abortion notwithstanding infamy the widow could have resorted to commit suicide by consumption of poison and which is beginning of all the events. Not many deaths consequent to criminal abortion are reported. Biswas H reported a case of 28 year old widow, who died due to perforation of uterus due to unskilled abortionist⁽⁶⁾. On the other hand the treatment summary do not mention of signs of disseminated intravascular coagulation³ or bleeding per vagina etc. The CA report was positive for the poison even after 4 days of indoor treatment for poisoning. This proves consumption of poison by the deceased. The

signs of abortion in the form of decidual reaction, is the motive for her to attempt to end her life by consuming poison to avoid infamy.

Prior to abortion Act (1967) in England, there occurred 30 deaths from criminal abortion each year. In 20 of these cases the abortionist was the patient herself⁽⁷⁾. By assessment of menstruation, destruction of embryos is deliberately done. The diagnosis of pregnancy from menstrual calculations is an ethical as well as legal query as pregnancy starts from the time of impregnation of zygote into the endometrium and interruption of normal pregnancy without indication contravenes both ethics and law⁽⁸⁾⁽⁹⁾.

Vital organic causes for suicides may prove as a starting point for police investigations. Though attempting a suicide is an offence, but other ancillary offences related to suicide like abetment of suicide may need a vital clue for further investigations.

Conclusion

Role of autopsy surgeon does not end with mere determining identity, know the cause of death, time since death, mode & manner of death or opinion about live born or dead born fetus, but every morbid anatomical findings in the dead body do reveal the disease or the condition that the person lived with, which indicate not only cause & manner but also motive of crime.

References:

1. Reddy KSR. Medicolegal autopsy. Essentials Forensic Med. Toxicol. 29th ed. Hyderabad: K.Suguna Devi; 2010. p. 93.
2. Parikh CK, Prabhakar PE. Text book of Medical Juris Prudence and toxicology. Mumbai: CBS publishers & distributors; 1992. p. 465.
3. Azim A, Begume N. Septic Abortion a major problem in Bangladesh. Int. Conf. Obstet. Gynaecol. 1992. p. 197–203.
4. Accidental deaths and suicidal deaths 2012 [Internet] <http://ncrb.nic.in> [Accessed on 26/03/2014].
5. Cunningham FG et al. Implantation, embryogenesis and placental development. William's Obstet. 22nd ed. New York: Mcgraw Hill; p. 49–52.
6. Biswas H, Das RK, Talukder SI. Death of a Case of Criminal Abortion by an Unskilled Abortionist. Dinajpur Med Col J. 2012;5(1):72–5.
7. Jeffcoate N, Tindal VR. Principles of Gynaecology. 5th ed. London: Buterworth and Co. Ltd; 1987. p. 624.
8. Islam A. MCH Programme in Bangladesh. Int. Conf. Obstet. Gynaecol. 1992. p. 53.
9. Nandi A. Principles of Forensic Medicine. 1st ed. New Central Book Agency; 1995. p. 395.

Review Article

HONEY BEE STINGS AND ANAPHYLAXIS: REVIEW

Dr. AP Rayamane, Dr. MP Kumar, Dr DG Kishor, Dr. Dayananda R, Dr. A Saraf

Authors:

Dr. Anand P Rayamane, Assistant Professor, Department of Forensic Medicine, Mysore Medical College and Research Institute, Mysore

Dr. M P Kumar, Associate Professor, Department of Forensic Medicine, Mysore Medical College and Research Institute, Mysore

Dr. D G Kishor, Resident, Department of Forensic Medicine, Mysore Medical College and Research Institute, Mysore

Dr. Dayananda R Assistant Prof, Department of Forensic Medicine, Mysore Medical College and Research Institute, Mysore

Dr. Ashish Saraf, Resident, Department of Forensic Medicine, Mysore Medical College and Research Institute, Mysore

Number of pages: Seven

Number of Tables: Nil

Number of Photographs: Two

Address for Correspondence:

Dr Anand P Rayamane, MD (FM) PGDMLE, PGDHR
Assistant Professor, Department of Forensic Medicine
Mysore Medical College and Research Institute,
Mysore-570001
07259114490
anandprayamane@gmail.com

Review Article

HONEY BEE STINGS AND ANAPHYLAXIS: REVIEW

Dr. AP Rayamane, Dr. MP Kumar, Dr DG Kishor, Dr. Dayananda R, Dr. A Saraf

Abstract:

Arthropod bites and stings are capable of inflicting injury, allergic reactions and transmitting infectious diseases. Hymenoptera order members are particularly important because of being nearly ubiquitous in the nature. Their stings may lead to fatal allergic reactions. The severity and duration of reaction to bee venom can differ from one person to another. Most people experience a local non-serious allergic reaction to bee venom. However, depending on the location and number of bee stings, previous history of allergic reactions may increase possibility of severe life-threatening events. Hymenoptera venom consists of a mixture of biologically active substances including enzymes which causes localized and systemic reactions which may be fatal. Diagnostic test are helpful in identifying allergy to venom and also to differentiate between bee, wasp, yellow jackets or hornets. Postmortem findings in bee stings are those of local reactions and internally upper respiratory tract edema found in anaphylaxis cases apart from systemic findings due to shock and venom allergens in treated cases. Absence of these finding doesn't rule out possibility of anaphylaxis.

Key words: Honey bee sting, Anaphylaxis, Autopsy.

Introduction:

Anaphylactic shock is an unexpected, sudden and sometimes deadly event that affects the patient in 75% of the cases without pre-existent history of allergy. According to the recent concept most common causes are drugs, hymenopterics poisons and nutrients.¹

The severity and duration of reaction to bee venom can differ from one person to another. Most people experience a local non-serious allergic reaction to bee venom. However, depending on the location and number of bee stings, previous history of allergic reactions may increase possibility of severe life-threatening events.

Insects of the order Hymenoptera, which include honey bees, wasps, ants and hornets, are frequently involved in accidental stings to human beings around the globe. Hymenoptera venom consists of a mixture of biologically active substances including enzymes which causes localized and systemic reactions which may be fatal.

Case Report:

The deceased was a 70 year male, who sustained accidental, multiple honey bee stings while he was grazing cows in field. He was taken to local hospital where stings were removed, first aid given and referred to higher centre. He died on his way to tertiary centre. On examination his face, neck and upper limbs were swollen. Multiple punctured stings with oozing of straw color fluid were observed around eyes (Fig-1). In-situ dissection of upper airway (Fig-2) revealed severe edema of tracheal and laryngeal mucosa. The thickness of this edema was comparable to the width of little finger. On cut section straw color fluid oozed out. Uvula and pharyngeal mucosa was edematous. Lungs were edematous and congested. Cause of death was ascertained as asphyxia due to upper airway edema in a case of multiple honey bee stings (Anaphylactic shock).



Fig. 1: Showing Multiple honey bee stings with oozing of straw color fluid.

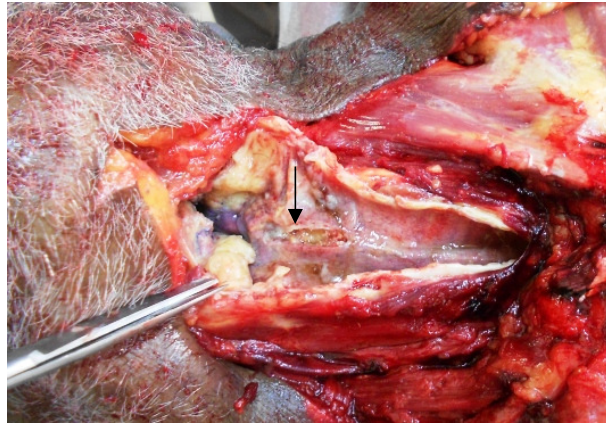


Fig. 2: In-situ dissection showing edema of laryngeal and tracheas mucosa with oozing of fluid.

Discussion and review:

Entomology: Arthropod bites and stings are capable of inflicting injury, allergic reactions and transmitting infectious diseases. Hymenoptera order members are particularly important because of being nearly ubiquitous in the nature. Their stings may lead to fatal allergic reactions².

The well-known members of the Hymenoptera order are bees, wasps, hornets, yellow jackets and ants. This order in fact consists of about 100,000 species of bees, wasps and ants. Many of these animals have poison glands and stinging apparatus²⁻⁵. Two distinct families exist, the Apidae and the Vespidae. The genus *Apis* contains only the honeybee, while the genus *Polistes* and the genus *Vespula* contain wasp, yellow jacket and hornet (Fig 1). The various subspecies occur with different frequencies in different parts of the world.⁶

Among the species of Hymenoptera, bees are the most commonly encountered species because of beekeeping activities⁷. Two of the more commonly encountered species of bee are honeybee *Apis mellifera* and the bumblebee (*Bombus* species). The venom of *Apis mellifera* (honey bee) extensively used in oriental medicine. Honey bee venom has both analgesic and anti-inflammatory effects. Therefore, it has been used for a variety of conditions, including pain syndrome, herniated nucleus pulposus, cervical disc protrusion, and progressive muscle atrophy.⁸⁻¹⁰

Pathogenesis: Bees sting only in defence, and during a sting approximately 50 µg of venom is injected into the skin. The barbed sting is normally left in situ, resulting in evisceration and death of the bee. Bees venom contains three main allergens: Phospholipase A (to which most patients are sensitive), hyaluronidase (to which a smaller number are allergic) and mellitin (which is an important allergen in only a few patients)⁶.

The non-allergic local reaction is a toxic response to venom constituents, while the large local reaction appears to be caused by an allergic reaction to venom proteins. The IgE-mediated late-phase reaction is probably responsible for most of these reactions; however, a cell-mediated mechanism, or a combination of the two, is possible.¹¹

Mellitin, a peptide component of bee venom, hydrolyses cell membranes, changes cell permeability, causes histamine and catecholamine release and is responsible for local pain. It acts with phospholipase-A2 to trigger the release of arachidonic acid, which causes cell membrane breakdown, damage of the vascular endothelium, and activation of the inflammatory response. Peptide 401 (mast cell degranulating peptide), triggers mast cells to degranulate, releasing histamine and other vasoactive peptides. Vasoactive amines, including

histamine, dopamine and noradrenaline can provoke ischemia and even myocardial infarction through profound hypotension and arrhythmia, or by increasing oxygen demands through direct inotropic and chronotropic effects in the presence of preexisting ischemic heart disease.¹²

The “allergic angina syndrome” which could progress to acute myocardial infarction (“allergic myocardial infarction”) was first described in 1991 by Kounis and Zavras. Allergic angina and allergic myocardial infarction are now referred to as “Kounis syndrome” this syndrome is associated with mast cell degranulation.¹³⁻¹⁵

Jae Woo Jung et al reported a fatal case of a 65-year-old woman with DIC (Disseminated intravascular Coagulation), following anaphylactic shock after bee sting acupuncture¹⁶.

Mesothelium damage, thrombocyte and macrophage activation, cytokine, leukotriene release vascular coagulation), bradykinin and Platelet activating factor (PAF) and sometimes even the deposition of immune complexes in the basement membrane of small blood vessels and activation of the complement system may contribute to the pathogenesis of DIC¹⁷.

Lethal dose and fatal period: The estimated lethal dose is approximately 20 stings/kg in most mammals. Anaphylactic reactions to Hymenoptera stings are not dose dependent or related to the number of stings. Onset of life-threatening, anaphylactic signs typically occur within 10 minutes of the stings¹⁸. Massive honey bee envenomation is defined as more than 50 stings at a time¹⁹.

Venom Allergens: Hymenoptera venom consists of a mixture of biologically active substances including enzymes (phospholipases, hyaluronidases), peptides (melitin, apamin, bombolitin) and other low molecular weight compounds (biogenic amines, acetylcholine, lipids and free amino acids)^{19,20}.

Clinical Features: Hymenoptera sting envenomation results in a number of clinical presentations (i) Non-allergic, local reactions (pain, minor edema, redness at the sting site); (ii) Allergic, large local reactions (extensive swelling >10 cm persisting more than 24hrs); (iii) Anaphylaxis (generalized urticaria, angioedema, bronchospasm, hypotension, cardiovascular collapse and loss of consciousness); (iv) Systemic toxic reactions (edema, vomiting, diarrhea, headache, seizures and altered sensorium); (v) Unusual reactions (cardiac ischemia, encephalomyelitis and cerebral infarctions).^{19,21,22}

Localized reactions: These vary in size from swellings a few centimetres in diameter to marked oedema of the entire hand, the forearm or even most of the leg.⁶ In most individuals hymenoptera stings cause nonallergic local reactions of limited size and duration. Some subjects experience an allergic local reaction (large local reaction) to a sting that is greater than 10 cm in diameter and may last for up to 5 days²³.

The most alarming localized reactions are those involving the eyelid or the subcutaneous tissues of the neck, although these are smaller than many of the swellings affecting the limbs. Whilst such reactions are frightening and may cause considerable discomfort, they are not dangerous. Large swellings take several days to subside⁶.

Systemic or generalized reaction: The commonest clinical features of the anaphylactic reaction to bee and wasp venom are cutaneous (pruritus, urticaria and angio-oedema) and respiratory (asthma and laryngeal oedema). Features particular to insect sting allergy are gastrointestinal symptoms (diarrhoea, abdominal pain and incontinence) and visual problems,

including transient amblyopia. Patients may also suffer from tachycardia, sweating, hypotension, fainting and loss of consciousness. In some patients a severe generalized reaction can occur very rapidly, sometimes within a few minutes of the sting. Many patients suffer from a sensation of impending doom⁶.

Hymenoptera antigen, when injected, can cause a wide range of severe delayed effects like serum sickness, neurological disturbances like polyradiculomyelitis (Guillain- Barre syndrome) and seizures, acute renal failure, haemolysis, thrombotic thrombocytopenic purpura (TTP), disseminated intravascular coagulation (DIC), myocardial infarction or cardiac arrhythmias. Atypical and unusual reactions were similarly seen in this case, some of which occurred a few hours or even a few days after the bee stings^{12, 24-26}.

Song et al.²⁷ and Cho et al.²⁸ reported a case of serum sickness reaction caused by honeybee acupuncture, and there was one report of fatal anaphylaxis to a bee sting after sensitization through repeated honeybee acupuncture²⁹. Zhang Ling et al³⁰ reported a case of multiple organ dysfunction due to massive bee stings in which diagnosis was based on autopsy findings of acute pulmonary edema, acute kidney injury, hepatic and cardiac dysfunction, and cerebral edema. Acute myocardial ischemia, severe rhabdomyolysis and angioedema following massive bee envenomation was also reported³¹.

Diagnostic tests: Diagnosis is based on history, skin tests and estimation of venom specific serum IgE-antibodies (RAST)³². To diagnose insect venom allergy a good patient history is important. Allergological tests (skin test, specific IgE titre) confirm the diagnosis³³.

Investigations are of value to confirm the clinical impression, and can be helpful to determine whether the allergy is to bee or wasp venom, if the insect has not been identified. skin-prick tests to the venoms and measurement of specific IgE antibodies in the serum are helpful in such conditions⁶.

Specific IgE (CAP-FEIA), CAST-ELISA (leukocyte sulfidoleukotriene release) and Flow-CAST (basophil CD63 expression) are valuable additional diagnostic tools for establishing the true culprit insect in patients with unclear clinical history or sensitization to both insects³⁴.

A serum tryptase and specific IgE to bee venom on serum obtained at autopsy can assist in confirming anaphylactic reaction to bee venom as the cause of death, particularly in the absence of observable stings^{12, 35}.

Treatment:

Symptomatic: The majority of Hymenopteran stings are self-limiting events, which resolve in a few hours without treatment. Treatment of uncomplicated envenomations (stings) consists of conservative therapy with antihistamines, ice or cool compresses, topical lidocaine, or corticosteroid lotions¹⁸.

Epinephrine is the only effective drug in case of respiratory (bronchial asthma, laryngeal edema) or cardiovascular (hypotension, arrhythmias, hypovolemic shock) manifestation. It has to be administered as rapidly as possible¹.

Immunotherapy: For individuals with a specific allergy to Hymenoptera venom, immunotherapy may be a relatively safe and effective treatment option². Immunotherapy leads to complete protection in more than 98% of patients with wasp (yellow jacket) venom allergy and in 75-80% of patients with bee venom allergy³³.

The efficacy of venom immunotherapy is well documented but this treatment is expensive. It is therefore mainly indicated in patients with a history of severe systemic reactions and a high degree of exposure^{32, 36, 37}.

Autopsy Findings: Multiple puncture mark over body with surrounding edema is found in death due to bee stings. On squeezing straw color edematous fluid oozes in fresh cases. There is swelling of whole limbs, face and neck. Stings are found in-situ, if not treated. Dead honey bees may be found on body.

In anaphylaxis there may be laryngeal (or pharyngeal, or other upper airway) oedema. Pulmonary oedema, if present, may indicate epinephrine (adrenaline) overdose. Recommended blocks for histological examination are: Heart, Coronary artery, Lung with airways, Vocal cord mucosa. Anaphylactic shock is likely to be misdiagnosed as myocardial infarction. Myocardial ischaemia is very probable (almost inevitable) in shock deaths, which may therefore be mistaken for primary myocardial infarction. Mode of death in anaphylaxis is asphyxia and respiratory arrest due to upper airway edema and Cause of death is anaphylactic shock³⁸.

Nezih Anolay et al³⁹ observed pulmonary edema and swelling in laryngeal area in a case of honey bee sting. In many cases of fatal anaphylaxis no specific macroscopic findings are present at postmortem examination. This reflects the rapidity and mode of death, which is often the result of shock rather than asphyxia. Investigations that might help determine whether anaphylaxis was the cause of death had rarely been performed. In the presence of a typical clinical history, absence of postmortem findings does not exclude the diagnosis of anaphylaxis⁴⁰.

Conclusion:

Honey bee stings most commonly occurs accidentally in rural population in fields. Honey bee keepers allergic to bee venom are advised to carry epinephrine or undergo immunotherapy by pure venom extract. In deaths due to Honey bee stings early postmortem and in-situ dissection of larynx and trachea will detect upper airway edema which will help to conclude cause of death as anaphylactic shock.

References:

1. Kaeser P, Hammann C, Luthi F, Enrico JF. Anaphylactic shock. Praxis (Bern) 1994; 1995 Nov 7;84(45):1307-13.
2. Steen CJ, Janniger CK, Schutzer SE, Schwartz RA. Insect sting reactions to bees, wasps, and ants. Int J Dermatol. 2005;44(2):91-4.
3. Johanson B, Eriksson A, Irnehult L. Human fatalities caused by wasp and bee stings in Sweden. Int J Legal Med. 1991;104(2):99-103.
4. Klotz JH, Klotz SA, Pinnas JL. Animal Bites And Stings With Anaphylactic Potential. J Emerg Med. 2009; 36(2):148-56.
5. Langley RL. Animal Bites and Stings Reported by United States Poison Control Centers, 2001-2005. Wilderness Environ Med. 2008;19(1):7-14.
6. Pamela W Ewan. Allergy to insect stings: a review. Journal of the Royal Society of Medicine. 1985;78
7. Allergy to wasp and bee stings. www.allergyuk.org. Accessed on 28/03/14 .
8. Lee JD, Park HJ, Chae Y, Lim S. An overview of bee venom acupuncture in the treatment of arthritis. Evid Based Complement Alternat Med 2005;2:79-84.
9. Kim HW, Kwon YB, Han HJ, Yang IS, Beitz AJ, Lee JH. Antinociceptive mechanisms associated with diluted bee venom acupuncture (apipuncture) in the rat formalin test: involvement of descending adrenergic and serotonergic pathways. Pharmacol Res 2005;51: 183-8.

10. Wesselius T et al. A randomized crossover study of bee sting therapy for multiple sclerosis. *Neurology* 2005;65: 1764-8.
11. Wright DN, Lockey RF. Local reactions to stinging insects (Hymenoptera). *Allergy Proc.* 1990 Jan-Feb;11(1):23-8.
12. Riches KJ, Gillis D, James RA. An autopsy approach to bee sting-related deaths. *Pathology* 2002 Jun;34:257-262
13. Zavras GM, Papadaki PJ, Kokkinis CE, Kalokairinov K, Kouni SN, Batsolaki M et al. Kounis syndrome secondary to allergic reactions following shellfish ingestion. *Int J Clin Pract.* 2003; 57(7):622-4.
14. Kounis GN, Hahalis G, Soufres GD, Kounis NG. Kounis syndrome and simultaneous multivessel acute coronary syndrome after drug eluting stent implantation. *Int J Cardiol.* 2008;127(1):146-8.
15. Kogias JS, Sideris SK, Anifadis SK. Kounis syndrome associated with hypersensitivity to hymenoptera stings. *Int J Cardiol.* 2007; 114(2):252-5.
16. Jae Woo Jung et. al. *Allergy Asthma Immunol Res.* 2012 March;4(2):107-109.
17. Severino M, Bonadonna P, Passalacqua G. Large local reactions from stinging insects from epidemiology to management. *Current Opinion in Allergy & Clinical Immunology.* 2009; 9:334-337.
18. Fitzgerald KT, Flood AA. Hymenoptera stings. *Clin Tech Small Anim Pract.* 2006 Nov;21(4):194-204.
19. Betten DP, Richardson WH, Tong TC, Clark RF. Massive honey bee envenomation induced rhabdomyolysis in an adolescent. *Paediatrics.* 2006;117:231-235
20. Habermann E. Bee and wasp venoms. *Science* 1972;177:314-22.
21. Levine HD. Acute myocardial infarction following wasp sting. *Am Heart J.* 1976; 91:365.
22. Brasher GW, Sanchez SA. Reversible electrocardiographic changes associated with wasp sting anaphylaxis. *JAMA.* 1974; 229: 1210-1
23. Wright DN, Lockey RF. Local reactions to stinging insects (Hymenoptera). *Allergy Proc.* 1990 Jan-Feb;11(1):23-8.
24. Knight B. The Pathology of Sudden Death. In: Saukko P, Knight B. *Knight's Forensic Pathology.* 3rd ed. London: Arnold, 2004:527-541.
25. Vij K. Sudden and Unexpected Deaths. In: *Text Book of Forensic Medicine and Toxicology.* 3rd ed. New Delhi: Elsevier, 2005: 197-215.
26. Barss P. Renal failure and death after multiple stings in Papua New Guinea. Ecology, prevention and management of attacks by vespid wasps. *Med J Aust* 1989; 151: 659-663.
27. Song HJ, Suh YJ, Yang YM, Jung JW, Lee YM, Suh CH, Nahm DH, Park HS. Two cases of anaphylaxis due to bee venom acupuncture. *J Asthma Allergy Clin Immunol* 2002;22:481-6.
28. Cho HJ, Choi GS, Kim JH, Sung JM, Ye YM, Park HS. A case of serum sickness reaction caused by honeybee acupuncture. *Korean J Asthma Allergy Clin Immunol* 2010;30:325-8.
29. Lee SH et al. A fatal case of bee venom anaphylaxis to bee sting after repeated honeybee acupuncture. *Korean J Asthma Allergy Clin Immunol* 2008;28:313-6.)
30. Multiple organ dysfunction syndrome due to massive wasp stings: an autopsy case report ZHANG Ling, TANG Yi, LIU Fang, SHI Yu-ying, CAO Yu, XU Huan and FU Ping
31. Anu Mathew, Anugrah Chrispal, Thambu David. Acute Myocardial Injury and Rhabdomyolysis caused by Multiple Bee Stings. *JAPI.* August 2011: 19
32. *Schweiz Med Wochenschr.* Clinical aspects, diagnosis and therapy of insect bite allergy. Müller U 1989 Dec 9;119(49):1761-8

33. Knulst AC, de Maat-Bleeker F, Bruijnzeel-Koomen CA. Wasp and bee venom allergy. *Ned Tijdschr Geneeskd.* 1998 Apr 18;142(16):889-92
34. Scherer K, Weber JM, Jermann TM, Krautheim A, Tas E, Ueberschlag EV, Cammarata M, Bircher J. Cellular in vitro assays in the diagnosis of Hymenoptera venom allergy. *AJInt Arch Allergy Immunol.* 2008;146(2):122-32. doi: 10.1159/000113515. Epub 2008 Jan 18.
35. Ansari MQ, Zamora JL, Lipscomb MF. Postmortem diagnosis of acute anaphylaxis by serum tryptase analysis. A case report. *Am J Clin Pathol* 1993; 99:101-3.
36. Przybilla B, Ruëff F. Desensitization of allergy to hymenoptera venoms. *Wien Med Wochenschr.* 1999;149(14-15):421-8
37. Tracy JM. Insect allergy. *Mt Sinai J Med.* 2011 Sep-Oct;78(5):773-83. doi: 10.1002/msj.20286.
38. Dr Emyr Benbow, Professor Sebastian Lucas, Dr Shuaib Nasser, Dr Richard Pumphrey and Professor Ian Roberts. Guidelines on Autopsy Practice Scenario 4: Autopsy for suspected acute anaphylaxis (includes anaphylactic shock and anaphylactic asthma), 2nd ed. April 2012; 2005 RCPATH Working Party on the Autopsy. 2012 amendments.
39. Nezhil Anolay, M. Nihat Arslan, Bahadır Kumral, Yalçın Büyük. Medicine science case report: Death Caused by Honey Bee Stings: Case Report.
40. Pumphrey RS, Roberts IS. Postmortem findings after fatal anaphylactic reactions. *J Clin Pathol* 2000; 53:273-6.