ORIGINAL ARTICLE

# Morphological and Histopathological Changes in Kidney and Suprarenal Glands in Hanging and Sudden Death – An Autopsy Based Study

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## ABSTRACT

***Background:*** Suicide by hanging mode is fourth leading cause of death; the majority in low and middle-income countries. Stress plays a key role in structural and functional changes in suprarenal glands and kidneys. ***Objective:*** To identify morphological and histopathological changes in kidney and suprarenal gland in hanging and correlating those changes with acute or chronic stress. ***Methods:*** This cross-sectional, descriptive study was conducted on 108 cases of hanging and sudden death collected from the Department of Forensic & State Medicine of a medical college in India, based on inclusion and exclusion criteria. All macroscopic and microscopic changes in kidneys and suprarenal glands were recorded. ***Results:*** Most of the cases belonged to the 21–40 years age group; a male predominance was observed and most of the cases were from the rural areas. The length of kidney, cortical thickness, tubular necrosis decrease in chronic stress but increase in acute. Surface haemorrhage, congested interstitium, congested glomerulus increase in chronic and acute stress but cloudy degeneration, tubular cast, interstitial oedema decrease in chronic and acute stress. Suprarenal gland weight, thickness, capsular haemorrhage, congestion, sinusoidal dilation, lipid depletion increase in chronic and acute stress, but necrosis decrease. Nodular hyperplasia decrease in chronic stress but increase in acute. Oedema decreased in acute stress but increased in chronic stress. ***Conclusion:*** Differences in morphological and histopathological changes in kidney and suprarenal gland were observed in acute and chronic stress conditions.

**Keywords:** Kidney, suprarenal gland, stress, hanging, sudden death, autopsy.

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## INTRODUCTION

Suicide is a complex global public health issue.[1] According to the World Health Organization (WHO) 2000-2019 estimates, almost eight lac people die every year due to suicide across the world.[2,3] Most of the victims are middle aged and post-marital deaths are more common than premarital deaths. Stress plays an important role in affecting adrenal gland anatomy and structure, which is partially regulated by adrenocorticotropic hormone (ACTH) stimulation, known to regulate stress corticosterone levels during the hypothalamo-pituitary-adrenal (HPA) axis activation due to an acute physical or psychological stressor.[3,4] Elevated plasma cortisol levels and adrenal enlargement have been found in individuals suffering from chronic stress and increased adrenal weight found in individuals who have committed suicide. Acute stress causes vascular changes and necrosis. Patients with

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depression have increased basal plasma cortisol and enlarged adrenals.[2-4] Furthermore, increased glucocorticoid levels have been linked with the onset and severity of depression, suggesting that alterations in peripheral HPA axis structure and function may also be clinically relevant.[3,4] The present work addresses the hypothesis that adrenal responses to ACTH are augmented after chronic stress and increased responsiveness is associated with increased weight, cortical hypertrophy and hyperplasia of adrenal glands.[4,5] Therefore, the present study was performed to observe differences in gross morphological and histopathological changes in the suprarenal glands and kidneys of people dying from hanging and from sudden death. We tried to identify morphological and histopathological adaptive changes in suprarenal glands and kidneys in hanging and in sudden death as well as correlate those changes with acute and chronic stress.

## METHODS

In this cross-sectional, descriptive study, a total of 108 cases of hanging and sudden death irrespective of age and gender were evaluated as collected from the mortuary of the Department of Forensic & State Medicine in a medical college in India. Cases brought for postmortem examination within 48 hours of death were included for study and those brought after 48 hours of death were excluded. Cases with doubtful, insufficient history and with previous history of chronic illness were also excluded. The study was based on purposive sampling method. Parameters of study were macroscopic and microscopic examination of collected specimens of kidneys and suprarenal glands. Current literatures were compiled by previous authors on this subject and corroborations done with the present study. The particulars of the deceased noted followed by collecting samples of kidney and suprarenal glands. The length, width and cortical thickness of kidneys, presence of gross congestion or punctate haemorrhages after removing perinephric fat and renal capsule was documented. Any other gross pathological findings like hydronephrosis, renal calculus or renal cyst and renal tumour were also noted. The length of the kidney was determined in mm by measuring the distance between the upper and lower poles using slide calipers, while the width measured the distance between hilum and the farthest point of lateral diameter of the respective kidney, following Johora et al.[6] The cortical thickness of the kidneys was measured by incising along the sagittal plane, following Siddiqua et al.[7] The suprarenal glands were examined grossly to note any abnormalities, surrounding structures, any obvious scars, haemorrhagic area or visible necrosis. Thickness from all dimensions and weight of the suprarenal glands were measured.[8,9] Selection of tissues hold a special significance for the histopathological examination from the diagnostic point of view. Here, 1″×1″×1″ blocks of kidney from the upper and lower poles were taken including cortex, medulla, renal pyramids with adequate glomeruli made. In both adrenal glands, 1″×1″×1″ blocks of tissues were taken. Histopathological techniques were adopted with preparation of tissues for the purpose of microscopic examination, which was accomplished by submitting total or selected part of tissues for examination to fixation, grossing, dehydration, clearing, impregnation, embedding, slicing by microtome and staining with haematoxylin and eosin (H&E) stain.

Data entry was done eventually following complete compilation of relevant data for a given subject. Statistical evaluation was done by appropriate statistical method using SPSS software version 29.0 for Windows (IBM, USA). Results were presented as mean±SD (standard deviation) for continuous variables and frequency and percentage for categorical variables. Chi-square test or Fisher's exact test were performed for estimating differences between variables. Statistical significance was considered, if p-value was found <0.05.

## RESULTS

The majority of cases in the study population (54 cases) were in 21–40 years age group, followed by 17 cases in 10–20 years, 28 in 41–60 years and rest nine belong to 61–80 years age group. Mean age was 35.37±14.98 years. The minimum and maximum age in the study population were 15 and 76 years respectively. 71 cases were male, while the rest 37 were female. 42 cases were Muslims and the rest 66 were Hindus. 73 cases were married and the rest 35 were unmarried. 67 cases resided in the rural areas and 41 were from the urban areas. The morphological and histopathological parameters of kidney and suprarenal glands associated with presence or absence of acute and chronic stress were correlated based on retrieved data and eliciting

history from relatives of deceased. 61.11% (66 cases) had history of acute stress whereas rest 38.89% (42 cases) did not have it. 60.19% (65 cases) had history of chronic stress whereas rest 39.81% (43 cases) did not have it.

The histopathological features showed evidence of acute and chronic stress in kidneys like surface haemorrhage, tubular cast, necrosis, congested glomerulus, oedema, cloudy degeneration and congested interstitium. Similar evidence was observed in suprarenal glands like capsular haemorrhage, congestion, nodular hyperplasia, sinusoidal dilation, necrosis, oedema and lipid depletion (Table 1-4). The length of kidneys, cortical thickness, tubular necrosis showed negative correlation with history of chronic stress but positive correlation in acute stress. Surface haemorrhage, congested interstitium, congested glomerulus showed positive correlation with history of chronic and acute stress, but cloudy degeneration, tubular cast, interstitial oedema showed negative correlation with history of chronic and acute stress. All the parameters in kidney showed no statistical significant difference with chronic and acute stress except congested glomeruli. (Table 5; Fig.1,2).

Similarly, thickness, weight, capsular haemorrhage, congestion, sinusoidal dilation, lipid depletion of the suprarenal glands showed positive correlation with history of chronic and acute stress. Necrosis of the glands showed negative correlation with history of chronic and acute stress. Nodular hyperplasia of the suprarenal glands showed negative correlation with history of chronic stress, but positive correlation and statistically significant difference with history of acute stress. Oedema of the glands showed positive correlation with history of chronic stress, but negative correlation with acute stress (Table 6; Fig. 3).

**Table 1:** Kidneys in acute stress

| Histological features | Category | Present | Absent | Total |
|---|---|---|---|---|
| Surface haemorrhage | Present | 41 | 25 | 66 |
| | Absent | 24 | 18 | 42 |
| Tubular cast | Present | 32 | 34 | 66 |
| | Absent | 23 | 19 | 42 |
| Tubular necrosis | Present | 45 | 21 | 66 |
| | Absent | 25 | 17 | 42 |
| Congested glomerulus | Present | 48 | 16 | 64 |
| | Absent | 21 | 21 | 42 |
| Oedema | Present | 34 | 32 | 66 |
| | Absent | 22 | 20 | 42 |

**Table 2:** Kidneys in chronic stress

| Histological features | Category | Present | Absent | Total |
|---|---|---|---|---|
| Surface haemorrhage | Present | 39 | 26 | 65 |
| | Absent | 26 | 17 | 43 |
| Cloudy degeneration | Present | 34 | 31 | 65 |
| | Absent | 25 | 18 | 43 |
| Congested interstitium | Present | 42 | 23 | 65 |
| | Absent | 31 | 12 | 43 |

**Table 3:** Suprarenal glands in acute stress

| Histological features | Category | Present | Absent | Total |
|---|---|---|---|---|
| Necrosis | Present | 05 | 61 | 66 |
| | Absent | 04 | 38 | 42 |
| Oedema | Present | 17 | 49 | 66 |
| | Absent | 12 | 32 | 43 |
| Lipid depletion | Present | 61 | 05 | 66 |
| | Absent | 38 | 04 | 42 |

**Table 4:** Suprarenal glands in chronic stress

| Histological features | Category | Present | Absent | Total |
|---|---|---|---|---|
| Capsular hemorrhage | Present | 60 | 05 | 65 |
| | Absent | 37 | 06 | 43 |
| Congestion | Present | 35 | 30 | 65 |
| | Absent | 22 | 21 | 43 |
| Nodular hyperplasia | Present | 38 | 27 | 65 |
| | Absent | 28 | 15 | 43 |
| Sinusoidal dilation | Present | 64 | 01 | 65 |
| | Absent | 41 | 02 | 43 |

**Table 5:** Statistical comparison between different parameters of kidney in acute and chronic stress

| Parameters | Laterality | Pearson's coefficient | | P-value | | Comment (statistic) |
|---|---|---|---|---|---|---|
| | | Acute stress | Chronic stress | Acute stress | Chronic stress | |
| Length | Right | +0.11 | -0.0102 | 0.26 | 0.916923 | Insignificant |
| | Left | +0.0138 | -0.00535 | 0.15 | 0.96 | Insignificant |
| Cortical thickness | Right | +0.16 | -0.12 | 0.09 | 0.21 | Insignificant |
| | Left | +0.11 | -0.16 | 0.25 | 0.1 | Insignificant |
| Surface haemorrhage | Right | +0.026 | +0.09 | 0.13 | 0.29 | Insignificant |
| | Left | +0.75 | +0.95 | 0.08 | 0.92 | Insignificant |
| Cloudy degeneration | Right | -0.064 | -0.24 | 0.5 | 0.94 | Insignificant |
| | Left | -0.14 | -0,042 | 0.42 | 0.66 | Insignificant |
| Tubular cast | Right | -0.26 | -0.06 | 0.91 | 0.53 | Insignificant |
| | Left | -0.004 | -0.13 | 0.97 | 0.95 | Insignificant |
| Tubular necrosis | Right | +0.09 | +0.23 | 0.36 | 0.08 | Insignificant |
| | Left | -0.42 | -0.005 | 0.94 | 0.96 | Insignificant |
| Congested interstitium | Right | +0.08 | +0.004 | 0.58 | 0.66 | Insignificant |
| | Left | +0.016 | +0.25 | 0.87 | 0.09 | Insignificant |
| Congested glomeruli | Right | +0.27 | +0.25 | 0.0047 | 0.0042 | Significant |
| | Left | +0.015 | +0.19 | 0.96 | 0.85 | Insignificant |
| Interstitial oedema | Right | -0.009 | -0.24 | 0.93 | 0.42 | Insignificant |
| | Left | -0.85 | -0.0266 | 0.21 | 0.78 | Insignificant |

**Table 6:** Statistical comparison between different parameters of suprarenal gland in acute and chronic stress

| Parameter | Laterality | Pearson's coefficient | | P-value | | Comment (statistical) |
|---|---|---|---|---|---|---|
| | | Acute stress | Chronic stress | Acute stress | Chronic stress | |
| Thickness | Left | +0.019 | +0.08 | 0.85 | 0.9 | Insignificant |
| | Right | +0.026 | +0.088 | 0.79 | 0.366 | Insignificant |
| Weight | Left | +0.0022 | +0.011 | 0.98 | 0.91 | Insignificant |
| | Right | +0.004 | +0.025 | 0.96 | 0.8 | Insignificant |
| Capsular haemorrhage | | +0.045 | +0.1 | 0.64 | 0.3 | Insignificant |
| Congestion | | +0.006 | +0.026 | 0.94 | 0.78 | Insignificant |
| Necrosis | | -0.034 | -0.097 | 0.72 | 0.32 | Insignificant |
| Nodular hyperplasia | | -0.25 | -0.067 | 0.001 | 0.5 | Insignificant |
| Oedema | | -0.03 | +0.11 | 0.75 | 0.26 | Insignificant |
| Sinusoidal dilation | | +0.045 | +0.04 | 0.64 | 0.67 | Insignificant |
| Lipid depletion | | +0.034 | +0.0285 | 0.72 | 0.77 | Insignificant |

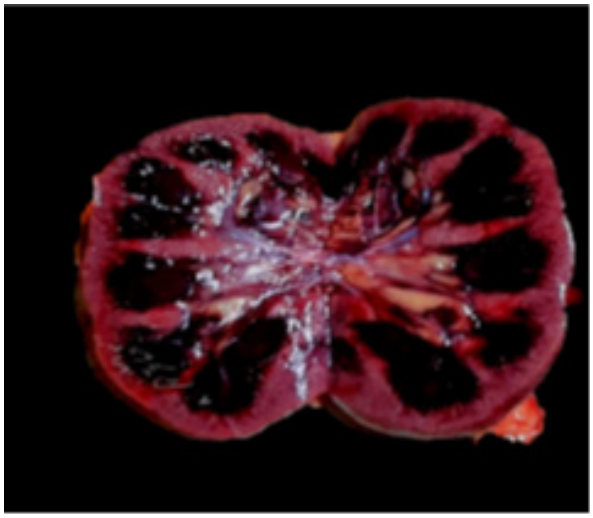

**Figure 1:** Morphological examination of the cut surface of kidney showing haemorrhage.

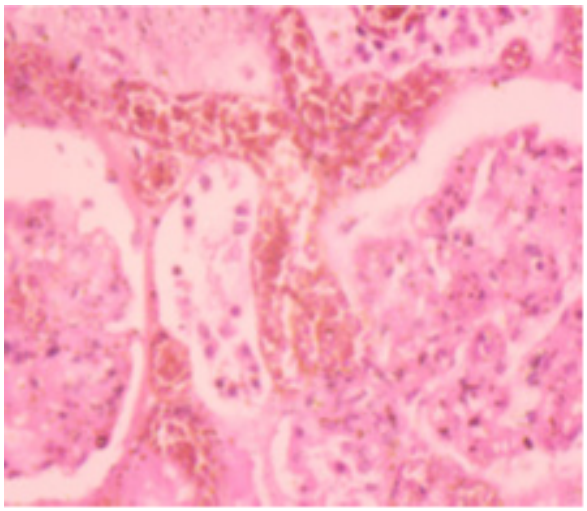

**Figure 2:** Histopathological examination of kidney showing congested interstitium (H&E stain; ×10 magnification).

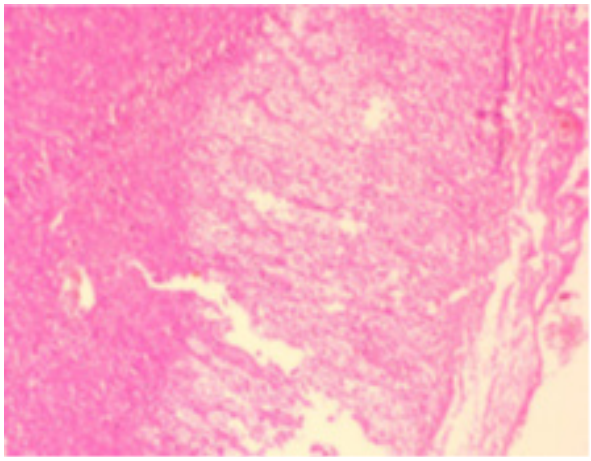

**Figure 3:** Histopathological examination of suprarenal gland showing sinusoidal dilatation (H&E stain; ×10 magnification).

## DISCUSSION

The most prevalent age group was 41–60 years (25.93%). The mean age of study population was 35.37±14.98 years ranging from 15–76 years. Vishwakarma et al. found that the 21–30 years age group is the most vulnerable for suicidal hanging (36.63% deaths occurred), followed by 31–40 years (23.84% deaths occurred).[10] Several other studies found that 21–30 years is the most common age group committing suicide by hanging,[10-13] while Mishra et al. found 30–39 years age group as the most common age group committing suicide by hanging.[14] The mean percentage of male and female population in present study was 65.74% and 34.26% respectively. Vishwakarma et al. found 77.91% male and 22.09% female among 172 cases.[10] Similar findings were observed in several other studies.[14-17] However, a contrast finding was observed by Ahmad et al.[18] The present study resemble study of Baishya et al., who found 88.66% of victims were Hindu.[13] Vishwakarma et al. found 7.56% cases were married and 42.44% unmarried.[10] However, Chavan et al. reported that unmarried cases outnumbered the married cases.[19] Our findings are similar to that of Gouda et al., who found 70.6% cases were from rural region and 29.4% from urban areas,[20] but dissimilar with that of Vishwakarma et al., who found urban areas outnumbered the rural area.[10] Mishra et al. reported 85.84% of study population belonged to urban region and only 14.16 % cases were from rural areas.[14] Similarly, Kumar et al. found 95.04 % of their study population were from urban region and 4.95% cases from rural area.[21]

Chakrabarti et al. found negative correlation between post stress survival duration and kidney length, cortical thickness and surface haemorrhage, which support the results of the present study.[22] According to Kadavkar et al. cloudy degeneration, tubular casts in renal tubules were common findings in cases with any duration of survival.[23] In contrast, Shinde & Keoliya showed tubular cast in 31.82% cases indicating acute kidney injury.[24] Sevitt et al. found renal tubular necrosis in 59% cases and acute stress had strong association with diffuse tubular necrosis.[25] Chakrabarti et al. found interstitial congestion lead to hypoxia in tubules further worsening the degree of tubular necrosis.[22] Sathikumar et al. found glomerular congestion

and hypoperfusion induced interstitial edema in kidney in their study.[26] Chakrabarti et al. also observed histopathologically evident interstitial oedema.[22]

Szigethy et al. found positive correlation between adrenal weight and total cortical thickness of both adrenal gland providing direct evidence that increased adrenal weight is due to cortical hypertrophy.[27] According to Sarkar et al., the total cortical width of both adrenal glands were significantly higher for suicide group than the control group. There was no significant difference in width of zona glomerulosa and zona reticularis between suicide group and the control group.[5] A study done by Willenberg et al., on morphological changes in adrenals from victims of suicide found a significant enlargement of adrenal cortex to 158.8% that was restricted to the two inner zones only.[28]

Manivannan et al. compared both adrenal thickness in suicidal death cases age-wise. There was significant difference in both adrenal in the 20–30 years age group (P<0.001) and in 41–50 years age group (P<0.001).[29] Dorovini and Zis had done an investigation on the expanded adrenal gland in casualties of suicide and adrenal gland weight was significantly higher in victims of violent suicide than those who died suddenly from an accidental cause.[30] These results bear a resemblance with present study when analyzed individually. Manivannan et al. found significant difference in the weight of left adrenal in age group of 31-40 years when compared to weight of the right adrenal.[29] Dumser et al. showed adrenal gland weight of victim can be a morphologic sign of depressive disorder prior to death if no other disease with known effect on the adrenals is present.[31]

Ulrich-lai et al. found increased adrenal weight under chronic stress conditions.[32] Chaurasia et al. found weight of left adrenal gland more than right in the suicidal group, but this difference in weight was not statistically significant (p=0.55).[33] However, they found 7%, 8%, 32%, and 16% of cases showing haemorrhage in the zona glomerulosa, fasciculata, reticularis, and medulla respectively, with none in controls.[33] Symington et al. described similar changes in severe fatal burns, occurring as early as 24 hours after injury and persisting for up to 10 days.[34] Hence, caution should be exercised in using these changes to differentiate chronically stressed adrenal glands from those exposed to the acute stress of dying.[34]

Chaurasia et al. graded the external appearance of adrenal gland from 1 to 4, indicating pale, normal, congested and haemorrhagic respectively. Of the 200 adrenal glands, 60% of cases were graded 4; 37% with grade 3; 2% graded 1; 1% were graded 2, and 1% appeared normal. Among the 20 controls, none exhibited haemorrhagic appearance or grade 4.[33] As both the findings of Chaurasia et al. and the present study corroborate with each other, it can be stated that congestion of adrenal gland more commonly seen in acute and chronic stress. However, Chaurasia et al. also stated that adrenal haemorrhage and necrosis associated with suicidal case appears to be specific to chronic stress condition.[33] They also stated that 48% of suicidal group and 20% of control group exhibited nodularity. Nodular hyperplasia as a criterion for histological diagnosis of chronically stressed adrenal glands may be imprudent. In contrast to the present study, Chaurasia et al. found the notable histological changes of focal lipid depletion in the zona fasciculata and reticularis, capsular haemorrhage, nodular hyperplasia, haemorrhage and necrosis along with oedema evident in acute stress.[33]

Symington et al. observed focal lipid depletion in acute stress and diffuse or complete lipid depletion in chronic stress.[34] Sarason suggest variations in intracellular lipids as a reactive change in the adrenal cortex under stressful conditions.[35] Sayers noted that lipid depletion indicates adrenal gland hyperactivity, as seen in stressed conditions.[36] Stoner et al. observed completely lipid-depleted adrenal in cases of chronic prolonged stress.[37] Similarly, Willenberg et al. and Patra et al. noted mild, moderate, and extensive grades of lipid depletion in suicidal cases, with no depletion in control cases.[28,38] These findings resemble that of the present study.

## CONCLUSION

Our data revealed a spectrum of histopathological events in kidneys and suprarenal glands in acute and chronic stress, which can enrich the idea of stress related structural and functional changes in

particular tissues in situ.

**Conflicts of Interest:** None declared by the authors.

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**Ethical Clearance:** Ethical clearance was granted by the Institutional Ethics Committee of Burdwan Medical College, West Bengal, India (Ref: BMC/ I.E.C./558; Dated 24.11.2022).

**Author's Contribution:** All authors were equally involved in conception, study design, data collection, statistical analysis, manuscript writing, editing and final submission.

## REFERENCES

1. World Health Organization (WHO). Suicide worldwide in 2019: global health estimates. Geneva: WHO; 2021.
2. Fazel S, Runeson B. Suicide. N Engl J Med. 2020;382:266-74.
3. Naghavi M; Global Burden of Disease Self-Harm Collaborators. Global, regional, and national burden of suicide mortality 1990 to 2016: systematic analysis for the Global Burden of Disease Study 2016. BMJ. 2019;364:l94.
4. Wu Y, Schwebel DC, Huang Y, Ning P, Cheng P, Hu G. Sex-specific and age-specific suicide mortality by method in 58 countries between 2000 and 2015. Inj Prev. 2021;27(1):61-70.
5. Sarkar A, Chatterjee M, Batabyal S. A postmortem study on the weight and morphology of adrenal glands in victims of suicide. Int J Curr Res Rev. 2014;6(1):21.
6. Johora F, Nurunnabi ASM, Siddiqua D, Hena H, Ara S. Morphometric study of the size of the postmortem human kidney. J Bangladesh Soc Physiol. 2020;15(2):61-7.
7. Siddiqua D, Nurunnabi ASM, Johora F, Hena H, Shahriah S, Ara S. Histomorphometric study of the proportion of cortex and medulla of the adrenal gland – a postmortem study. Mediscope. 2021;8(1):1-6.
8. Siddiqua D, Nurunnabi ASM, Ara S, Johora F, Hena H, Pervin S. A postmortem study on the volume of the human adrenal glands. Bangladesh J Med Sci. 2013;12(3):282-5.
9. Siddiqua D, Ara S, Nurunnabi ASM, Ahmed R, Perven HA. A postmortem study on the weight of the human adrenal glands. Bangladesh J Med Sci. 2010;9(4):204-7.
10. Vishwakarma AK, Thakur PS, Singh BK, Singh BK, Jain AP, Pendro TS, et al. An autopsy based study about epidemiological profile of deaths due to hanging in Central India Region, Madhya Pradesh. J Indian Acad Forensic Med. 2021;43(4):349-53.
11. Abouhashem AA, Bataw SM, Hegazy NI, Ibrahim OY. Suicidal, homicidal and accidental hanging: comparative cross sectional study in Aljabal Alakhdar area, Libya. Zagazig J Forensic Med Toxicol. 2020;18(1):126-39.
12. Ahmad M, Hossain MZ. Hanging as a method of suicide: retrospective analysis of postmortem cases. J Armed Forces Med Coll Bangladesh. 2010;6(2):37-9.
13. Baishya MK, Mahanta P. An epidemiological study of hanging cases brought to the Gauhati Medical College and Hospital mortuary for medico legal autopsy – a retrospective study. Medico-Legal Update. 2014;14(2):128-33.
14. Mishra PK, Tomar JS, Varun A, Verma P. Profile of deaths due to hanging – an autopsy based retrospective study at a tertiary care center in Indore. Indian J Forensic Comm Med. 2018;5(1):34-8.
15. Sharma BR, Harish D, Singh VP, Singh P. Ligature mark on neck: how Informative? J Indian Acad Forensic Med. 2005;27(1):10-5.
16. Badkur DS, Yadav J, Arora A, Bajpayee R, Dubey BP. Nomenclature for knot position in hanging: a study of 200 cases. J Indian Acad Forensic Med. 2012;34(1):34-6.
17. Sharija S, Sreekumari K, Geetha O. Epidemiological profile of suicide by hanging in southern parts of Kerala: an autopsy based study. J Indian Acad Forensic Med. 2011;33(3):237-40.
18. Ahmad M, Rahman FN, Hussain MA, Chowdhury MH, Yasmeen BHN. A medico legal study of hanging cases at Dhaka Medical College. Northern Int Med Coll J. 2015;7(1):110-4.
19. Chavan BS, Singh GP, Kaur J, Kochar R. Psychological autopsy of 101 suicide cases from northwest region of India. Indian J Psychiatry. 2008;50(1):34-8.
20. Gouda MRN, Rao SM. Factors related to attempted suicide in Davanagere. Indian J Comm Med. 2008;33(1):15-8.
21. Kumar C, Rana N, Goyal AK, Tanna J, Pathak A, Patil VR. Epidemiology of cases of hanging at Vadodara, Gujrat. Int Res Pub Med Sci. 2015;1(2):23-6.
22. Chakrabarti N, Banerjee U, Bandyopadhyay A, Das A, Roy DG. Estimation of post burn survival duration – a predictive equation model and a new perspective with

reference to detailed histomorphological changes of kidney in fatal burn injuries – an autopsy based study in a tertiary teaching hospital of Eastern India. Int J Contemp Med Res. 2017;4(1):279-83.

23. Kadavkar SM, Bansude ME, Umbare RB, Dode CR. A histopathological study of vital organs in deaths due to burns. J Indian Acad Forensic Med. 2022;44(2):78-85.
24. Shinde AB, Keoliya AN. Study of burn deaths with special reference to histopathology in India. Indian J Basic Appl Med Res. 2013;2(8):1153-9.
25. Sevitt S. Distal tubular and proximal tubular necrosis in the kidneys of burned patients. J Clin Pathol. 1956;9(4):279-94.
26. Sathikumar M. Study of histopathological changes in lungs and kidneys following death due to burns at varying periods of survival. J Evol Med Dent Sci. 2016;5(61):4301-4.
27. Szigethy E, Conwell Y, Forbes NT, Cox C, Caine ED. Adrenal weight and morphology in victims of completed suicide. Biol Psychiatry. 1994;36(6):374-80.
28. Willenberg HS, Bornstein SR, Dumser T, Ehrhart-Bornstein M, Barocka A, Chrousos GP, et al. Morphological changes in adrenals from victims of suicide in relation to altered apoptosis. Endocr Res. 1998;24(3-4):963-7.
29. Manivannan K, Hussain K, Rao HK, Sasikumar R, Reddy DR, Raman PJ. The morphometric analysis of human adrenal glands of hanging and poison suicidal death cases in different age group. Int J Curr Res Rev. 2021;13(08):41.
30. Dorovini-Zis K, Zis AP. Increased adrenal weight in victims of violent suicide. Am J Psychiatry. 1987;144(9):1214-5.
31. Dumser T, Barocka A, Schubert E. Weight of adrenal glands may be increased in persons who commit suicide. Am J Forensic Med Pathol. 1998;19(1):72-6.
32. Ulrich-Lai YM, Figueiredo HF, Ostrander MM, Choi DC, Engeland WC, Herman JP. Chronic stress induces adrenal hyperplasia and hypertrophy in a subregion-specific manner. Am J Physiol Endocrinol Metab. 2006;291(5):E965-73.
33. Chaurasia S, Trivedi A, Ganvir R, Singh S, Yadav J, Malik R, et al. Histological changes in adrenal glands in suicidal and sudden death cases: a postmortem study. Cureus. 2024;16(2):e54630.
34. Symington T, Currie AR, Curran RC, Davidson JN. The reaction of the adrenal cortex in conditions of stress. In: Ciba Foundation Colloquia on Endocrinology. Vol. VIII. London: J&A Churchill Ltd.; 1955.
35. Sarason EL. Adrenal cortex in systemic disease: a morphologic study. JAMA Intern Med. 1943;71(5):702-12.
36. Sayers G. The adrenal cortex and homoestasis. Physiol Rev. 1950;30:241-320.
37. Stoner HB, Whiteley HJ, Emery JL. The effect of systemic disease on the adrenal cortex of the child. J Pathol Bacteriol. 1953;66:171-83.
38. Patra S, Rath S, Dutta BK, Mohapatra C, Mishra GB, Sahu RK, et al. Histological study of adrenal gland in case of suicidal deaths. IOSR J Dent Med Sci. 2014;13(8):30-6.