Original Research Article

A histopathological study of visceral organs in death due to burns

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ABSTRACT

Background: Burns is a burning problem perhaps ever since human being existence particularly, from its use of fire. Now-a-days due to increasing industrialization and urbanization, we are facing this problem in greater magnitude. Earlier studies described burns as a silent epidemic. Besides causing death in some cases, it is the cause of lifelong disfigurement, deformity and disability of the body. Major burn-injury is also the cause of psychological disturbance and financial loss to the individual/ victim and also the family and society at large. Our aim is to examine histopathology in different organ and skin affected due to burn.

Materials and Methods: A cross-sectional descriptive study was conducted over a period from February 1988 to November 1989. Study of cytomorphological changes in visceral organs particularly brain, heart, lung, liver, spleen, kidney and adrenal in cases of death due to burns, received at autopsy section of Forensic Medicine and Toxicology department, Government Medical College, Aurangabad, Maharashtra.

Results: In our study, burn due to domestic accidents found in 83.80%. The inflammatory material that was instrumental in causing burn injury was found as kerosene in 90(69.23%), cloths worn (flames) in 35 (26.92%) cases, diesel in 2 cases (01.53%), gasoline, petrol scalding in 1 (0.76%) case each; we found that burn were caused by inflammable liquids in 71.20% cases. Based on history and dying declaration, nature of burn injury was assumed as accidental in 110 (84.62%) cases, suicidal in 19 (14.06%) cases and homicidal in 1 (0.76%) cases.

Conclusion: Present study shows that most of the cases died within 7 days of sustaining burns. Microscopic examination of visceral organs such as brain, heart, lung, liver, spleen, kidney and adrenal were carried out and morphological changes were observed in each organ. Microscopic observations were recorded and analyzed keeping in view the time sequence after burn incidence. Congestion of various organs was seen in all cases which died within first 24hours.

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1. Introduction

Burns is a burning problem perhaps ever since human being existence particularly from its use of fire.¹ Now-a-days due to increasing industrialization and urbanization, we are facing this problem in greater magnitude. Previous studies described burns as a silent epidemic. Besides causing death in some cases, it is the cause of lifelong disfigurement, deformity and disability of the body. Major burn-injury is also the cause of psychological disturbance and financial loss to the individual/ victim and also the family and society

at large. Fire constitutes the third leading cause of death in U.S.A. Burns constitute a major cause of death and the morbidity in India. Accidental, suicidal and homicidal deaths due to burns are all common in India.² Burns are occurring in all circumstances of human- life i.e. domestic, peridomestic, industrial, environmental etc. In India burns due to domestic accidents are most common and are also causing the most exhaustive and severe injuries to the body. Mortality due to burns is related with so many factors e.g. age, sex, severity of burns, nature of the agent causing burns, time of the burn injury, time interval between injury and treatment etc. considering the low socio- economic status

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of women in India. They are more vulnerable to sustain the burn injury. Social problems like dowry, illiteracy, ignorance etc. contribute for female preponderance in burn occurrence. Females are set on fire for financial gains, for suspicions of chastity or infidelity. Although it is difficult to decide the exact cause of death in most of cases of burns an attempt has been made by various authors to determine roles of various factors like sepsis, acute renal failure, burns shock, neurogenic shock etc in causation of death in case of burn injury. It is well known fact that all organs are affected due to burns. But most commonly the cytomorphological changes are seen in brain, heart, lungs, liver, spleen, kidney and adrenals.³

2. Materials and Methods

A cross-sectional descriptive study was conducted over a period from February 1988 to November 1989. Study of cytomorphological changes in visceral organs particularly brain, heart, lung, liver, spleen, kidney and adrenal in cases of death due to burns, received at autopsy section of Forensic Medicine and Toxicology department, Government Medical College, Aurangabad, Maharashtra. The study was carried out prior to 1993 i.e. in the 1988-89, when Institutional Ethics Committee Clearance wasn't required / mandatory. This work includes the study of 130 cases of burns admitted during the period from February 1988 to November 1989. There were no specific selection of cases but specimen were obtained from those consecutive autopsy cases of death due to burns, on whom the autopsy was performed within 6 hours of death. On admission extent of body surface affected due to burns was calculated as per rules of nine. Classification of burns was done according to Wilson as epidermal, dermo-epidermal and deep burns. The detailed clinical examination and necessary investigation of the patients were carried out according to the proforma.

All the specimens were preserved in 10% formalin for fixation. In histo-pathological examination, number of sections were taken from different sites according to size of specimen. Then these sections were processed. After processing the section were embedded in paraffin, cut with microtome at 5 mm thickness and stained with Hematoxylin and Eosin (H&E). Special staining procedure like Periodic Acid Schiff (PAS) was done as and when required.

All relevant data recorded, were analyzed by using Statistical Package for the Social Sciences (SPSS) software and the results were calculated in the form of frequency and percentage.

3. Observations

There were total 130 cases, out of which 35 were male and 95 were female.

In this study the minimum age was 1 year and maximum was 75 years. Religion wise distribution of the subject is as

follows.

The Table 1 and 2 showed that 15 cases were unmarried and rest 115 were married (11.54% and 88.46% respectively). Maximum cases were persons between age group of 21 to 40 yrs.⁴ (77 out of 130 - i.e. 59.24%).

According to the nature of burn injury (as per history given by patient or dying declaration). Survival period of maximum cases was between 1 to 7 days, 70 out of 130 (i.e. 53.85%).

Distribution of cases according to predominant factor as to the cause of death in Table 4.

4. Discussion

Out of 130 cases 15 (11.54%) were unmarried and rest 115(88.46%) were married. Majority of women were recently married and there is room to believe that burn incidents may be related to dowry related problems. The majority i.e. 105 out 130 was Hindus, 25 were Muslim and 2 being christen, represented the demographic distribution of population. Manner of sustaining the burns was studied and it was found that burns were due to bursting of stoves in 52(40%) cases, soaking clothes with kerosene in 18 (13.84%), fall of chimney (Kerosene lamp) on body. Saree catching fire from while warming body in 9 (06.92%) cases, bursting of diesel tank in 2 (01.53%) cases, due to boiling water causing burns, pouring petrol on body (self), leakage of cooking gas causing fire, petromax bursting in 1 (00.76%) each. These findings are comparable to observations recorded by Forester and Richardson.⁵ Domestic burns due to cooking accidents were found in 62.29% of cases in our study and these are similar to the findings noted by Joanne G. parks et al.⁶ in their study as 57% cases. This is due to fact that the females are busy to prepare the food in home along with various other works. In our study, burn due to domestic accidents was found in 83.80%. The inflammatory material that was instrumental in causing burn injury was found as kerosene in 90(69.23%), cloths worn (flames) in 35 (26.92%) cases, diesel in 2 cases (01.53%), gasoline, petrol scalding in 1 (00.76%) case each; we found that burn were caused by inflammable liquids in 71.20% cases whereas Forester and Richardson found in 18.60% cases.

Based on history and dying declaration, nature of burn injury was assumed as accidental in 110 (84.62%) cases, suicidal in 19 (14.06%) cases and homicidal in 1 (00.76%) cases (Table 2). Bull had observed similar findings in accidental cases.⁷

Congestion of brain was observed in all the cases which succumbed to injury within 24 hours of sustaining burns. Petechial haemorrhages seen in few cases can be attributed to anoxia. Oedema was seen in few cases belonging to each group of survival period. Jackson et al had noted liquefaction necrosis of brain in their studies. No such necrosis was found in this study except degenerative

Table 1: Religion wise distribution of the subject

Religion	Male	Female	Total	Percentage (%)
Hindu	28	77	105	80.76
Muslim	06	17	23	17.60
Christian	01	01	02	01.53
Total	35	95	130	100

Table 2: Types of burn injury

Nature of burn Injury	No. of Cases	Percentage (%)
Accidental	110	84.62
Suicidal	19	14.62
Homicidal	01	00.76
Total	130	100

Table 3: Percentage of body surface involved in burn injury

Percentage of burn	No. of Cases	Percentage (%)
Upto 40	11	08.46
41 to 60	22	16.92
61 to 80	44	33.84
81 to 100	53	40.78
Total	130	100

Table 4: Predominant factors as to the cause of death

Cause of death	No. of Cases	Percentage (%)
Shock	27	20.77
Toxemia	30	23.08
Septicemia	34	26.15
Bronchopneumonia	11	08.46
Pneumonia	09	06.92
Renal Failure	14	10.77
Toxic myocarditis	05	03.85
Total	130	100

Table 5: Histopathological changes in relation to percentage (%) of burn injury are as below

Group	Total No. of Cases	Congestion	Oedema	Pneumonia	Broncho-Pneumonia	Haemorrhage	Desquamative Interstitial Pneumonia
21 to 40	11	08 72.72%	04 36.36%	02 18.18%	01 09.09%	–	03 27.27%
40 to 60	22	14 63.63%	11 50.00%	02 09.09%	06 27.27%	01 04.54%	06 27.27%
61 to 80	44	32 72.72%	20 25.45%	05 11.36%	04 09.09%	01 02.27%	11 25.00%
81 to 100	53	41 77.35%	20 37.73%	–	–	01 01.88%	10 18.80%
Total	130	95 73.08%	55 42.31%	09 06.92%	11 08.46%	03 02.31%	30 23.08%

Table 6: Histopathological changes in relation to cause of death in burn injury are as below

Cause of death	Total No. of Cases	Congestion	Oedema	Pneumonia	Broncho-Pneumonia	Haemorrhage	Desquamative Interstitial Pneumonia
Shock	27	27 100%	06 22.22%	–	–	–	–
Toxemia	30	29 96.66%	15 50.00%	–	–	–	07 23.33%
Septicaemia	34	25 73.52%	27 89.41%	–	–	01 02.94%	08 23.52%
Broncho Pneumonia	11	04 36.36%	04 36.36%	–	11 100.00%	01 09.09%	07 63.63%
Pneumonia	09	06 66.66%	–	09 100.00%	–	01 11.11%	07 77.77%
Renal failure	14	02 14.28%	02 14.28%	–	–	–	01 07.14%
Toxic myocarditis	05	02 40.00%	01 20.00%	–	–	–	–
Total	130	95 73.08%	55 42.31%	09 06.92%	11 08.46%	03 02.31%	30 23.08%

Table 7: Histopathological changes in relation to survival period in burn injury are as below

Survival period in days	Total No. of Cases	Congestion	Oedema	Pneumonia	Broncho-Pneumonia	Haemorrhage	Desquamative Interstitial Pneumonia
Up to 01	20	20 100.00%	05 25%	—	—	—	—
01 to 03	34	17 79.41%	13 38.23%	—	01 02.94%	—	08 23.52%
03 to 07	36	26 72.72%	20 55.55%	03 08.33%	02 05.55%	03 08.33%	09 25.00%
07 to 10	13	08 61.53%	05 38.46%	02 15.38%	02 15.38%	—	13 100.00%
10 and above	27	14 51.85%	12 44.44%	04 14.81%	06 22.22%	—	10 37.03%
Total	130	95 73.08%	55 42.31%	09 06.92%	11 08.46%	—	30 23.80%

Table 8: Histopathological changes in brain in relation to cause of death

Group	Total No of cases	Congestion	Degeneration	Oedema
Shock	27	26 (96.29%)	02(07.40%)	01 (03.70%)
Toxemia	30	27 (90.00%)	02(06.66%)	04 (13.32%)
Septicaemia	34	27 (19.41%)	03(08.82%)	08(23.52%)
Bronchopneumonia	11	03 (27.27%)	—	01(09.09%)
Pneumonia	09	06(66.66%)	—	—
Renal Failure	14	—	—	—
Toxic myocarditis	05	01(20.00%)	—	—
Total	130	90(69.23%)	07(05.38%)	14 (10.76%)

Table 9: Histopathological changes in brain in relation to survival period

Survival period in days	Total No. of Cases	Congestion	Degeneration	Oedema
Up to 01	20	20 (100.00%)	—	01 (05.00%)
01 to 03	34	28 (82.35%)	03(08.82%)	03 (08.82%)
03 to 07	36	21 (58.33%)	02(05.55%)	04(11.11%)
07 to 10	13	09 (69.23%)	—	01(07.69%)
10 and above	27	12 (44.44%)	02(07.40%)	05(18.51%)
Total	130	90 (69.23%)	07(05.38%)	14(10.76%)

Table 10: Histopathological changes in liver in relation to cause of death in burn injury are as below

Cause of death	Total No. of Cases	Congestion	Degeneration	Necrosis	Regeneration	Haemorrhage	Hepatic Sinusoidal Dilatation	Infective foci
Shock	27	21 77.77%	03 11.11%	05 18.51%	—	01 03.07%	—	—
Toxemia	30	19 63.33%	11 36.66%	14 46.66%	02 06.66%	—	04 13.32%	01 02.94%
Septicaemia	34	14 41.17%	08 23.52%	30 88.23%	04 11.76%	01 02.94%	08 23.52%	01 09.09%
Broncho Pneumonia	11	03 27.27%	01 09.09%	01 09.09%	—	—	02 18.18%	—
Pneumonia	09	05 55.55%	—	01 11.11%	—	—	01 11.11%	—
Renal failure	14	02 14.28%	02 14.28%	—	—	—	—	—
Toxic myocarditis	05	02 40.00%	—	—	—	—	—	—
Total	130	66 50.76%	25 19.23%	51 39.23%	06 04.60%	02 01.52%	16 12.30%	02 01.53%

Table 11: Histopathological changes in liver in relation to survival period in burn injury are as below

Survival period in days	Total No. of Cases	Congestion	Degeneration	Necrosis	Regeneration	Haemorrhage	Hepatic Sinusoidal Dilatation	Infective foci
Up to 01	20	20 100.00%	03 15.00%	01 05.00%	–	–	01 05.00%	–
01 to 03	34	17 50.00%	10 29.41%	15 44.11%	–	–	02 05.88%	–
03 to 07	36	13 36.11%	09 25.11%	17 47.22%	01 02.77%	01 02.77%	06 16.66%	02 02.55%
07 to 10	13	03 23.07%	–	06 46.14%	01 07.69%	–	01 07.69%	–
10 and above	27	13 44.18%	03 11.11%	12 44.44%	04 14.80%	–	06 22.22%	–
Total	130	66 50.76%	25 19.23%	51 39.23%	06 04.70%	01 00.76%	16 12.30%	02 01.53%

Table 12: Histopathological changes in spleen in relation to cause of death in burn injury are as below

Cause of death	Total No. of Cases	Congestion	Degeneration	Necrosis	Follicular Hyperplasia	Hyaline like material in arteriole	Haemorrhage	Acute Inflamm.
Shock	27	27 100.00%	03 11.11%	–	–	–	–	–
Toxemia	30	28 93.33%	05 16.66%	06 66.00%	–	–	–	–
Septicaemia	34	34 100.00%	06 17.66%	06 17.66%	01 02.94%	05 14.70%	02 05.88%	01 02.94%
Broncho Pneumonia	11	05 45.45%	01 09.90%	–	–	–	–	–
Pneumonia	09	05 55.55%	01 11.11%	–	–	–	–	–
Renal failure	14	03 21.42%	01 07.14%	01 07.14%	–	–	–	–
Toxic myocarditis	05	02 40.00%	–	01 20.00%	–	–	–	–
Total	130	104 80.00%	17 13.07%	10 06.69%	01 00.76%	05 03.84%	02 01.53%	01 00.76%

Table 13: Histopathological changes in spleen in relation to survival period in burn injury are as below

Survival period in days	Total No. of Cases	Congestion	Degeneration	Necrosis	Follicular Hyperplasia	Hyaline like material in arteriole	Haemorrhage	Acute Inflamm.
Up to 01	20	20 100.00%	02 10.00%	–	–	–	–	–
01 to 03	34	34 100.00%	17 64.00%	01 02.94%	–	01 02.94%	01 02.94%	–
03 to 07	36	27 75.00%	04 11.11%	04 11.11%	01 02.77%	01 02.77%	01 02.77%	–
07 to 10	13	09 69.73%	–	01 07.69%	–	01 07.69%	01 07.69%	01 07.69%
10 and above	27	14 51.85%	05 18.51%	04 14.81%	–	03 11.11%	–	–
Total	130	104 80.00%	17 13.07%	10 06.69%	01 00.76%	06 04.61%	03 02.30%	01 00.76%

changes in brain on 2nd day onwards in few cases.⁸

Congestion of heart was seen to 90% cases which died within first 24 hours of sustaining burns. It was also predominant finding in few cases which survived beyond 24 Hrs. Focal minimal interstitial myocarditis was observed in 5 (3.84%) cases. This could be due to toxins produced. Clark has stated metabolic toxin as a cause of myocarditis in 6% cases of burns. Teplitz mentioned that, the heart in burns showed no specific or significant primary cardiac lesion. He observed interstitial myocarditis in 08.80% cases. Ahauer

et al stated burn toxin which caused chemical injury to myocardium, as a cause of myocarditis.

Congestion of lung was seen in all cases which died within 24 hours of sustaining burns. Foley stated that congestion was frequently the only finding in patients with extensive who died in early post burn period.

In the present study oedema was found in 42.30% cases which is also comparable to those of other studies (Shook, 1969 (66%) Foley, 1969(30.7%) Teplitz, 1969 (32%) Stone et al 1969 (40.50%) and Pruitt et al (1970) 30.80%. Teplitz

Table 14: Histopathological changes in kidney in relation to cause of death in burn injury are as below

Cause of death	Total Number of Cases	Congestion	Degeneration	Necrosis	Casts RBC in tubule	Haemo-rrhage	Interstitial Nephritis
Shock	27	26 96.29%	02 07.40%	05 18.51%	—	—	—
Toxemia	30	20 66.66%	07 23.33%	11 36.66%	02 06.66%	02 06.66%	—
Septicaemia	34	17 50.00%	09 26.47%	23 67.64%	01 02.94%	02 05.89%	—
Broncho-Pneumonia	11	02 18.18%	04 36.36%	—	—	—	—
Pneumonia	09	05 55.55%	01 11.11%	01 11.11%	—	—	—
Renal failure	14	01 07.14%	—	03 21.42%	—	—	02 14.28%
Toxic myocarditis	05	02 40.00%	—	—	—	—	—
Total	130	73 56.15%	23 17.69%	43 33.07%	03 02.30%	04 03.07%	02 01.53%

Table 15: Histopathological changes in kidney in relation to survival period in burn injury are as below

Survival period in days	Total Number of Cases	Congestion	Degeneration	Necrosis	Casts RBC in tubule	Haemo-rrhage	Interstitial Nephritis
Up to 01	20	20 100.00%	01 05.00%	04 20.00%	—	—	—
01 to 03	34	26 76.47%	10 29.41%	08 23.52%	02 05.88%	02 05.88%	—
03 to 07	36	17 47.22%	03 08.33%	14 38.88%	01 02.77%	02 05.55%	—
07 to 10	13	04 30.76%	01 07.69%	06 46.15%	—	—	—
10 and above	27	06 22.22%	08 29.62%	11 40.74%	—	—	02 07.40%
Total	130	73 56.15%	23 17.69%	43 33.07%	03 02.30%	04 03.07%	02 01.53%

Table 16: Histopathological changes in adrenal in relation to cause of death in burn injury are as below

Cause of death	Total Number of Cases	Congestion	Degeneration	Necrosis	Haemo-rrhage	Fat depletion	Infective foci
Shock	27	22 81.48%	03 11.11%	09 33.33%	02 22.22%	05 18.51%	—
Toxemia	30	19 63.33%	07 23.33%	14 46.66%	03 10.00%	04 13.33%	01 03.33%
Septicaemia	34	21 61.76%	13 38.23%	21 61.76%	07 20.58%	09 26.47%	01 02.94%
Broncho-Pneumonia	11	04 36.36%	02 18.18%	01 09.09%	01 09.09%	02 18.18%	—
Pneumonia	09	04 44.44%	01 11.11%	02 22.22%	01 11.11%	—	—
Renal failure	14	03 21.42%	01 07.14%	—	—	—	—
Toxic myocarditis	05	02 40.00%	—	—	—	—	—
Total	130	75 57.69%	27 20.76%	47 36.15%	14 10.76%	20 15.38%	02 01.53%

Table 17: Histopathological changes in adrenal in relation to survival period in burn injury are as below

Survival period in days	Total Number of Cases	Congestion	Degeneration	Necrosis	Haemo-rrhage	Fat depletion	Infective foci
Up to 01	20	20 100.00%	01 05.00%	06 30.00%	—	05 25.00%	—
01 to 03	34	20 58.02%	09 26.47%	13 38.23%	03 08.82%	02 05.88%	02 05.88%
03 to 07	36	21 58.33%	05 13.88%	12 33.33%	08 22.22%	07 19.44%	—
07 to 10	13	04 30.76%	—	07 53.84%	—	—	—
10 and above	27	10 37.03%	12 44.44%	09 33.33%	03 11.11%	06 22.22%	—
Total	130	75 57.69%	27 20.76%	47 36.15%	14 10.76%	20 15.38%	02 01.53%

thought congestion and oedema as reflection of systemic circulatory failure rather than primary pulmonary insult. Foley (1969) considered it as an effect of acute inhalation injury. He strongly suggested that inhalation of toxic gases rather than thermal injury, was responsible for oedema. He blamed congestion and oedema as a cause of death.

In this study changes in liver were found as congestion 66 (50.76%) cases. Similar finding were noted by Artz et al (1979) was found in 50% cases. Jackson et al (1963) and Panke et al (1985) also quoted congestion and necrosis in liver in their studies.

The hepatic necrosis was found in 49.11% cases who survived up-to 3 days. Similar findings were noted by Wilson (1939), Teplitz (1939) as midzonal necrosis. These were also noted by Zink (1940). Regeneration was found in 04.60% cases in this study. This histological finding is consistent with appearance of large nuclei, with prominent nuclear membrane, bi-nucleation of hepatic cells with mitosis at places.^{8–10}

The predominant findings in spleen were congestion in 104(80%) cases followed by degeneration 17(13.07%), necrosis 10(06.69%), hyaline like material in arteriolar wall in 5 (03.84%), haemorrhage in 3 (02.30%) follicular hyperplasia and acute inflammation in 1 case.¹¹

Predominant findings in kidneys were congestion in 73 (56.15%), followed by necrosis 43 (33.07%) degeneration 23 (17.69%) haemorrhage 4 (03.07%) RBC casts in tubule in 3 (02.30%) and interstitial nephritis 2(01.53%) cases. Renal cortical necrosis was found in 33.07% cases.¹²

Congestion of adrenal gland 75 (57.69%) and was followed by necrosis in 47 (36.15%) and degeneration in 29 (20.76%) cases. Fat depletion was found in 20 (15.38%) cases.¹³

In this study almost all vital and major organs were included for histopathological examination. Though the number was sizeable, the study was dependent on available cases.

Simultaneously study from histo-chemistry point of view could have been carried out.

5. Summary & Conclusion

A prospective autopsy study is carried out in 130 cases of deaths due to burns from February 1988 to November 1989. Out of 130 cases 35 were males and 95 were females. 80.76% subject belonged to the Hindu religion. The manner of sustaining burns by bursting of stove was predominant etiological factor. Use of kerosene as accelerant was very common (69.23%). Based on history given by the patients, accidental burns were the most common, followed by suicidal and homicidal. The age group 21 to 40 years was involved in 59.24% cases. Most of the cases died within 7 days of sustaining burns.

91.54% cases sustained more than 40% Burns. Microscopic observations of visceral organs: brain, heart, lung,

liver, spleen, kidney and adrenal were recorded and analyzed keeping in view the time sequence after burn incidence. Congestion was seen in all cases which died within first 24 hours. On 2nd day onwards in brain, heart the degeneration and focal minimal interstitial myocarditis were observed subsequently. In lung oedema, bronchopneumonia, pneumonia and desquamative interstitial pneumonitis were important findings. In liver degenerative changes, necrosis, regeneration and hepatic sinusoidal dilatation were seen on 2nd day onwards. Similarly spleen showed degeneration necrosis, presence of hyaline like material in arteriolar walls and haemorrhage. In kidney degenerative changes and necrosis of tubular epithelium were observed from 1st day. Adrenal showed congestion, degeneration, necrosis, fat depletion and haemorrhage from 1st day onwards. However, it is very difficult to pin-point a cause of death, particularly a single one. It appears that combination of factors like neurogenic shock, hypovolemic shock, toxemia, infections and respiratory complications including presence of bronchopneumonia, pneumonia etc. along with pulmonary oedema, are responsible. Cerebral oedema, pulmonary oedema and generalized congestion also seem to be important in adversely affecting the life processes. In few cases myocarditis with degenerative changes may also contribute to death. In certain number of cases liver necrosis may play an important role by causing metabolic derangement in ultimate fatal result. Acute renal tubular necrosis with varying degree of degenerative changes in kidney also appear to be important contributory factors in causing death particularly in 3 to 7 days. Contrary to observations by many workers in earlier studies, present study shows significant and extensive changes in adrenal in form of congestion, fat depletion, haemorrhage and necrosis in various combinations.

6. Source of Funding

None.

7. Conflict of Interest

None.

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