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Original Research Article

Gross and Microscopic Autopsy Findings in SARS-Cov-2-Positive Decedents Identified Through Pre-Autopsy Screening: A Cross-Sectional Observational Study

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ABSTRACT

Background: SARS-CoV-2 positivity detected before medico-legal autopsy may coexist with clinically apparent disease or with deaths from unrelated causes. Systematic descriptions of gross and microscopic findings across several organs in such decedents are useful for forensic interpretation.
Objectives: To characterise demographic and clinical features, organ weights, gross autopsy changes and multi-organ histopathological findings among SARS-CoV-2-positive decedents.
Methods: A descriptive, cross-sectional autopsy study was conducted in the Departments of Forensic Medicine and Pathology, Government Medical College, Ernakulam, from January to December 2021. Thirty consecutive decedents with positive pre-autopsy SARS-CoV-2 screening were included. Frequencies and percentages were summarised for categorical findings, and adult organ weights and body mass index were described separately from the infant case.
Results: Of 30 decedents, 25 (83.3%) were male and 21 (70.0%) were classified as natural deaths. Eleven (36.7%) were symptomatic and 15 (50.0%) had at least one recorded comorbidity. Airway froth was present in 22 (73.3%), gross heavy lungs in 21 (70.0%), and pulmonary artery thrombi in seven (23.3%). Microscopically, hyaline membranes were recorded in 11 (36.7%), alveolar capillary microthrombi in 15 (50.0%), and acute tubular injury in 13 (43.3%). Microthrombi were also recorded in the kidney (9/30), liver (7/30), brain (7/30) and heart (3/30).
Conclusion: Pre-autopsy SARS-CoV-2-positive decedents showed heterogeneous pulmonary and extrapulmonary pathological findings, including haemorrhagic and thrombotic changes. Such findings require interpretation alongside the circumstances of death and comorbid conditions; screening positivity alone does not establish a COVID-19-related cause of death.

Keywords: Forensic Autopsy, SARS-Cov-2 Screening, Pulmonary Microthrombi, Organ Pathology, Post-Mortem Histopathology.

INTRODUCTION

Autopsy findings became central to understanding coronavirus disease 2019 (COVID-19) because respiratory failure, thromboembolic disease and pre-existing morbidity could produce overlapping clinical presentations. In forensic practice, the diagnostic challenge is broader: a positive pre-autopsy SARS-CoV-2 test may be discovered in someone who died from a natural disease or an unrelated external event. Consecutive autopsy observations have shown the value of complete post-mortem examination in characterising infection-associated changes.[1] Medicolegal experience has further emphasised that detecting the virus and assigning the cause of death are separate tasks.[2]

Early pathological investigations documented pulmonary congestion, oedema, hyaline membranes and alveolar epithelial injury in fatal COVID-19.[3] Subsequent post-mortem series also identified capillary congestion,

parenchymal damage and abnormalities in tissues beyond the respiratory system.[4] Vascular injury and alveolar microthrombosis received particular attention after comparative studies of fatal COVID-19 and influenza-related respiratory failure.[5] Pulmonary microangiopathy with haemorrhage was also reported in a dedicated lung-and-heart autopsy series.[6]

Yet these pathological findings are not exclusive to COVID-19. Haemorrhage, oedema, vascular thrombi and chronic organ changes can arise from agonal events, circulatory failure, other infections or comorbid conditions. A systematic review of post-mortem findings highlighted both the extent of possible multi-organ involvement and the variation in sampling and reporting across studies.[7] Indian conventional autopsy research has documented pulmonary, renal and vascular abnormalities in clinically fatal COVID-19,[8] while minimally invasive studies have characterised pulmonary and extrapulmonary tissue injury in hospital deaths.[9,10] Those clinical cohorts are not fully comparable with an autopsy population selected on pre-autopsy screening positivity.

The present investigation therefore described the demographic and clinical features, manner of death, organ weights, gross findings and microscopic pathological observations in decedents with SARS-CoV-2-positive pre-autopsy screening. Particular attention was given to pulmonary abnormalities, froth in the central airways and recorded histological findings in the lungs, heart, liver, kidneys, spleen and brain.

MATERIALS AND METHODS

Study Design, Setting and Duration

This descriptive cross-sectional observational study was conducted at the Department of Forensic Medicine in collaboration with the Department of Pathology, Government Medical College, Ernakulam, Kerala, India. The stated study period was January to December 2021. Autopsies were undertaken in a designated facility with COVID-19-related biosafety precautions.

Study Population and Eligibility

The study included 30 consecutive medico-legal autopsy cases with positive routine pre-autopsy SARS-CoV-2 screening. The protocol specified a post-mortem interval of less than 24 hours as an inclusion criterion. It excluded cases with advanced decomposition, destructive injuries preventing meaningful internal examination, inadequate or severely autolysed tissue, negative SARS-CoV-2 screening, or inability to undertake autopsy for administrative or biosafety reasons. The screening method specified nasopharyngeal sampling and reverse-transcription polymerase chain reaction (RT-PCR); all included records were documented as RT-PCR positive.

Autopsy and Histopathological Examination

External and internal examination was performed according to the departmental forensic autopsy procedure. Organ weights and descriptive findings were recorded, including lung congestion, oedema, consolidation, haemorrhage, pulmonary artery thrombi, pleural effusion, lung consistency, appearance of the cut surface and the presence of fine froth in the trachea and bronchi. Gross observations of the heart, liver, kidneys and spleen were also assessed. Separate gross morphological observations for the brain were unavailable, although brain weights were recorded.

The protocol described the collection of lung sections from multiple anatomical areas; cardiac sections from ventricular myocardium, septum and coronary arteries; hepatic and renal tissue sections; splenic specimens; and cerebral tissue when available. Tissues were fixed in 10% neutral buffered formalin for 48–72 hours and processed as formalin-fixed paraffin-embedded sections. Haematoxylin and eosin staining was used for routine histological examination, with special stains when indicated. The protocol specified independent examination by two pathologists blinded to the clinical cause of death.

Variables and Analytical Approach

Variables selected for analysis were age group, sex, body mass index (BMI), manner of death, symptomatic status, fever, cough, breathlessness, comorbidities, organ weights, recorded gross autopsy observations and organ-specific histopathological findings. BMI was calculated from height and weight after verification of individual recorded measurements. The three-month-old infant was described separately rather than classified using adult BMI thresholds or included in adult organ-weight summaries.

Categorical observations were expressed as number and percentage. Adult BMI and organ weights were summarised by mean ± standard deviation and range. Cross-tabulations of selected pulmonary histological features by symptomatic status were descriptive; no

hypothesis-testing p-values were presented because the cohort was small and symptom status overlapped with the manner of death. Repeated labels describing the same organ-level finding were counted once per case. Separately named, potentially overlapping alveolar haemorrhage fields were retained as recorded and were not assumed to identify mutually exclusive lesions.

RESULTS

Demographic and Clinical Profile

The study included 30 RT-PCR-positive decedents, comprising 25 males (83.3%) and five females (16.7%). There were 29 adults aged 20–75 years and one three-month-old male infant. Among adults, mean age was 50.6 ± 17.9 years, and mean BMI was 23.45 ± 3.19 kg/m². Natural death was recorded for 21 cases (70.0%), and nine (30.0%) were classified under specific unnatural-death circumstances. Eleven cases (36.7%) were symptomatic and 19 (63.3%) were recorded as asymptomatic (Figure 1). Hypertension and ischaemic heart disease were each documented in 10 cases (33.3%), and diabetes mellitus in two (6.7%); some decedents had more than one condition (Table 1).

Characteristic	n (%)
Age category	—
Infant (<1 year)	1 (3.3)
20-39 years	9 (30.0)
40-59 years	7 (23.3)
≥60 years	13 (43.3)
Male	25 (83.3)
Female	5 (16.7)
Symptomatic	11 (36.7)
Asymptomatic	19 (63.3)
Fever	3 (10.0)
Cough	5 (16.7)
Breathlessness	10 (33.3)
Any comorbidity	15 (50.0)
Hypertension	10 (33.3)
Ischaemic heart disease	10 (33.3)
Diabetes mellitus	2 (6.7)
Natural death	21 (70.0)
Unnatural death (total)	9 (30.0)
Hanging	5 (16.7)
Poisoning	1 (3.3)
Snakebite	1 (3.3)
Drowning	1 (3.3)
Accidental injury	1 (3.3)
Adult BMI category (n=29)	—
Underweight (<18.5 kg/m²)	1 (3.4)
Normal (18.5-24.9 kg/m²)	23 (79.3)
Overweight (25.0-29.9 kg/m²)	3 (10.3)
Obesity (≥30 kg/m²)	2 (6.9)

Table 1. Demographic and Clinical Characteristics (N=30; Adult BMI N=29; Overlapping Symptoms and Comorbidities)

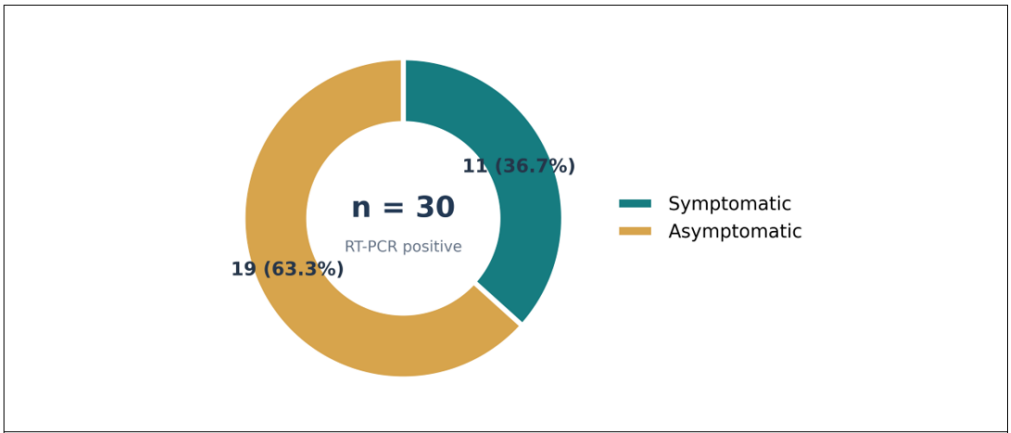


Figure 1. Recorded symptomatic status among SARS-CoV-2-positive autopsy cases

Donut chart shows 11 symptomatic and 19 asymptomatic decedents (n=30)

Organ Weights

Adult organ weights are summarised in Table 2. Mean right and left lung weights were 458.9 ± 75.6 g and 426.2 ± 73.3 g, respectively. Mean heart weight was 344.6 ± 41.6 g, and mean liver weight was 1483.3 ± 118.4 g. Organ weights for the infant were retained as a separate observation and not pooled with adult measurements.

Organ	Adult mean ± SD (n=29)	Adult range	Infant (n=1)
Right lung	458.9 ± 75.6	345-687	100
Left lung	426.2 ± 73.3	315-654	90
Total lung weight	885.1 ± 146.9	660-1341	190
Heart	344.6 ± 41.6	230-410	70
Brain	1287.9 ± 51.4	1200-1430	750
Liver	1483.3 ± 118.4	1200-1800	300
Right kidney	122.6 ± 18.0	80-145	30
Left kidney	116.9 ± 17.3	78-140	30
Spleen	112.6 ± 15.8	80-145	35

Table 2. Weights of Examined Organs at Autopsy (Grams)

The infant was three months old. Infant organ weights are individual observations and are not compared with adult reference ranges

Gross Post-Mortem Findings

Gross lung congestion and oedema were recorded in all 30 cases. Haemorrhage was documented in 22 (73.3%), heavy lungs in 21 (70.0%) and firm pulmonary consistency in 11 (36.7%). Seven cases (23.3%) had recorded pulmonary artery thrombi and seven (23.3%) had pleural effusion; consolidation was present in four (13.3%). Fine froth in the trachea and bronchi was observed in 22 cases (73.3%). These findings are detailed in Table 3 and Figure 2. Gross cardiac, hepatic, renal and splenic congestion were recorded in all cases. Coronary atheroma was identified macroscopically in 17 (56.7%) and gross fatty liver appearance in 25 (83.3%).

Organ	Gross finding	n (%)
Lungs	Congestion	30 (100.0)
Lungs	Oedema	30 (100.0)
Lungs	Haemorrhage	22 (73.3)
Lungs	Heavy lungs	21 (70.0)
Lungs	Firm consistency	11 (36.7)
Lungs	Dark-red to grey cut surface	20 (66.7)
Lungs	Consolidation	4 (13.3)
Lungs	Pulmonary artery thrombi	7 (23.3)
Lungs	Pleural effusion	7 (23.3)

Airways	Fine froth in trachea/bronchi	22 (73.3)
Heart	Congestion	30 (100.0)
Heart	Hypertrophy	3 (10.0)
Heart	Coronary atheroma	17 (56.7)
Heart	Thrombus in vessels	0 (0.0)
Liver	Congestion	30 (100.0)
Liver	Fatty appearance	25 (83.3)
Right kidney	Congestion	30 (100.0)
Left kidney	Congestion	30 (100.0)
Spleen	Congestion	30 (100.0)
Table 3. Selected Gross Autopsy Findings Across Major Organs (N=30)		
Congestion and oedema are non-specific post-mortem observations. No separate gross brain observations were available. Repeated lung-congestion fields were counted once per case		

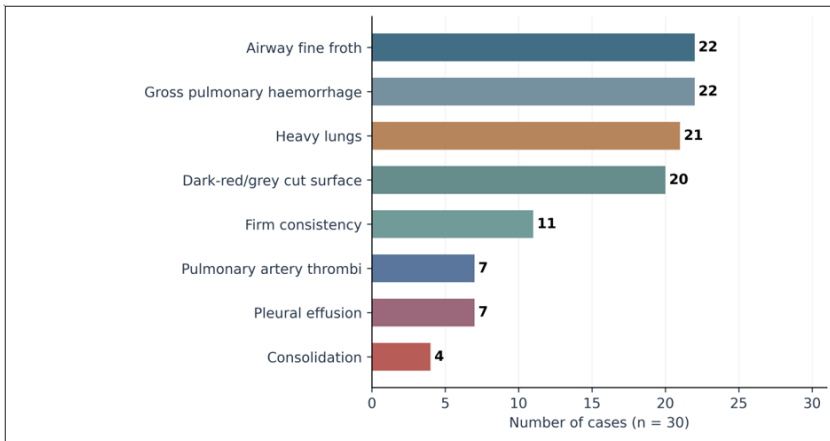


Figure 2. Frequency of selected gross pulmonary and airway findings

Bars show recorded numbers of cases with each feature; categories are not mutually exclusive (n=30)

Pulmonary Microscopic Findings

On microscopic examination, alveolar congestion and oedema were recorded in all 30 cases. Alveolar haemorrhage was recorded in 26 (86.7%), while a separate field labelled diffuse alveolar haemorrhage was positive in 21 (70.0%). Hyaline membranes were present in 11 (36.7%), type II pneumocyte hyperplasia in six (20.0%) and interstitial lymphocytic infiltrates in 12 (40.0%). Alveolar capillary microthrombi were identified in 15 (50.0%) and bronchopneumonia or secondary bacterial infection in seven (23.3%). Table 4 presents the full pulmonary findings. Selected features stratified by symptomatic status are illustrated descriptively in Figure 3.

Histopathological finding	**n (%)**
Diffuse alveolar haemorrhage	21 (70.0)
Hyaline membranes	11 (36.7)
Type II pneumocyte hyperplasia	6 (20.0)
Interstitial lymphocytic infiltrate	12 (40.0)
Alveolar oedema	30 (100.0)
Alveolar haemorrhage	26 (86.7)
Alveolar congestion	30 (100.0)
Alveolar capillary microthrombi	15 (50.0)
Bronchopneumonia/secondary infection	7 (23.3)
Table 4. Microscopic Pulmonary Findings (N=30)	
Diffuse alveolar haemorrhage and alveolar haemorrhage were separately recorded overlapping descriptors and were not merged. Staged diffuse alveolar damage was not available as a separate recorded diagnosis	

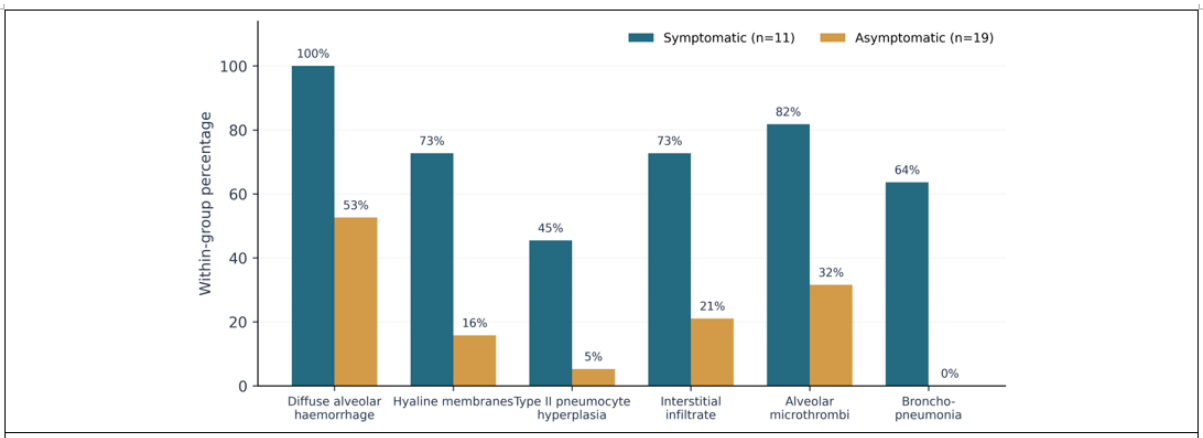


Figure 3. Selected pulmonary microscopic findings by recorded symptomatic status

Grouped bars show within-group percentages (symptomatic n=11; asymptomatic n=19). Comparisons are descriptive and are potentially confounded by manner of death

Extrapulmonary Microscopic Findings

In the heart, interstitial oedema/fibrosis was recorded in 16 cases (53.3%), myocarditis in five (16.7%), myocyte hypertrophy in four (13.3%) and microthrombi in three (10.0%). Hepatic congestion was recorded in all cases, steatosis in 23 (76.7%) and hepatic vascular microthrombi in seven (23.3%). Renal glomerular congestion was present in all cases, acute tubular injury in 13 (43.3%), interstitial inflammation in 12 (40.0%) and vascular microthrombi in nine (30.0%). Splenic congestion was recorded in every case. The recorded brain microscopic variables comprised congestion in all 30 cases, oedema in 19 (63.3%) and microthrombi in seven (23.3%); perivascular lymphocytic infiltration and meningitis were not recorded. Table 5 details these features and Figure 4 compares recorded microthrombi across organ systems.

Organ	Histopathological finding	n (%)
Heart	Myocyte hypertrophy	4 (13.3)
Heart	Interstitial oedema/fibrosis	16 (53.3)
Heart	Microthrombi	3 (10.0)
Heart	Myocarditis (recorded field)	5 (16.7)
Heart	Myocardial necrosis	0 (0.0)
Liver	Congestion	30 (100.0)
Liver	Steatosis	23 (76.7)
Liver	Vascular microthrombi	7 (23.3)
Kidney	Acute tubular injury	13 (43.3)
Kidney	Glomerular congestion	30 (100.0)
Kidney	Interstitial inflammation	12 (40.0)
Kidney	Vascular microthrombi	9 (30.0)
Spleen	Congestion	30 (100.0)
Brain	Oedema	19 (63.3)
Brain	Congestion	30 (100.0)
Brain	Microthrombi	7 (23.3)
Brain	Perivascular lymphocytic infiltrate	0 (0.0)
Brain	Meningitis	0 (0.0)

Table 5. Recorded Microscopic Findings In Extrapulmonary Organs (N=30)

The myocarditis category is reported as recorded and is not independently histologically adjudicated. Gross cerebral findings were unavailable; the table reports only recorded microscopic brain variables

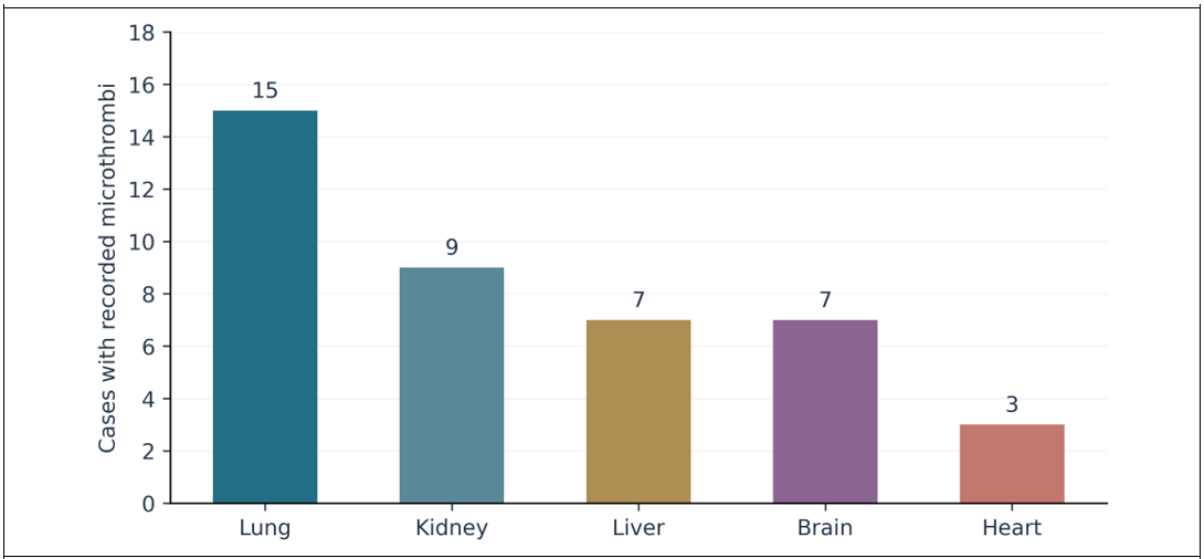


Figure 4. Microthrombi recorded on microscopic examination across organ systems

Organ-specific counts (n=30); presence in multiple organs was possible in the same decedent. No organ-specific viral causality is inferred

DISCUSSION

The principal observation in this autopsy series is that SARS-CoV-2 screening positivity occurred alongside a wide range of pulmonary and extra-pulmonary abnormalities, even when antecedent clinical symptoms were not recorded. The cases included natural and unnatural deaths and therefore differed from hospital autopsy series restricted to individuals who died of severe, clinically recognised COVID-19. Their heterogeneity is important. It increases the relevance of the work to forensic medicine, but makes the causal attribution of individual lesions to SARS-CoV-2 especially uncertain.

Pulmonary oedema and congestion were recorded across the whole cohort, while froth in the trachea and bronchi and gross pulmonary haemorrhage were common. These appearances are compatible with terminal pulmonary fluid accumulation and vascular disturbance, but neither airway froth nor congestion is specific for COVID-19. A more discriminating assessment relies on histological evidence interpreted with clinical circumstances, cause of death, treatment history and possible alternative mechanisms. In the Indian conventional autopsy study of Kyada and colleagues, diffuse alveolar damage, pulmonary thrombosis and acute tubular injury were notable pathological findings in fatal COVID-19.[8] Unlike that cohort, the present cases were identified through pre-autopsy screening rather than selection for COVID-19-associated mortality.

Hyaline membranes, pneumocyte hyperplasia and alveolar capillary microthrombi were recorded in a subset of cases, broadly reflecting lesion types described in early fatal-COVID investigations. The comparatively frequent microscopic haemorrhage requires cautious interpretation because two separately recorded fields distinguished alveolar haemorrhage and diffuse alveolar haemorrhage without a reproducible grading definition. Importantly, diffuse alveolar damage was not coded as a staged diagnosis in the analysed observations; its frequency or phases therefore cannot be inferred from the presence of hyaline membranes alone. Studies using systematic post-mortem histological sampling in India found diffuse alveolar damage in many patients who died of COVID-19, but their disease severity and sampling framework limit direct comparison.[9,10]

Pulmonary capillary microthrombi were documented in half the cases, with further microthrombi recorded in the kidney, liver, brain and heart. This distribution is compatible with the vascular pathology emphasised by Ackermann and colleagues,[5] yet microthrombi can also accompany hypoxia, systemic inflammation, disseminated coagulation abnormalities and other terminal conditions. The data cannot establish whether thrombi appeared simultaneously across organs or whether they were directly caused by infection. In a systematic review, Satturwar and colleagues reported thrombosis and haemorrhage among several post-mortem

abnormalities but stressed substantial heterogeneity between studies.[7]

An Indian clinical and virological autopsy study by Yadav and colleagues documented multi-organ pathology but found that organ-level viral detection did not always track closely with histological features.[11] Acute tubular injury was documented in 13 decedents, while glomerular congestion occurred in all cases. Such renal alterations deserve attention because they may reflect haemodynamic instability, comorbid vascular disease, tissue hypoxia or infection-related injury. Santoriello and colleagues described acute tubular pathology in a series of COVID-19 autopsies and considered multiple potential mechanisms for renal injury.[12] In this study, the absence of contemporaneous renal function measures and detailed agonal circumstances prevents attribution of individual renal abnormalities to COVID-19.

Cardiac findings pose a similar interpretive problem. Coronary atheroma was recorded macroscopically in 17 cases, and myocardial inflammatory changes were noted in five. However, the findings labelled myocarditis were not accompanied by available information on histological diagnostic thresholds, inflammatory-cell phenotype or exclusion of alternative causes. A review of 277 post-mortem examinations found histologically convincing myocarditis to be uncommon in COVID-19.[13] The current frequency should therefore be regarded as a recorded histopathology category rather than a verified estimate of definitive SARS-CoV-2 myocarditis.

Congestion, oedema and vascular lesions were also noted in brain tissue fields, although separate gross cerebral observations were not available. Solomon and colleagues described predominantly hypoxic and vascular-related neuropathological changes in fatal COVID-19, illustrating the need to distinguish a terminal physiological response from direct viral neuropathology.[14] The multi-organ picture here also included frequent hepatic steatosis and congestion, findings that may arise from pre-existing metabolic disease, circulatory disturbances or critical illness. Broader systematic reviews have similarly shown that many extrapulmonary abnormalities lack specificity for SARS-CoV-2.[15]

Several features of the study are relevant to Indian medico-legal practice. Routine pre-autopsy RT-PCR screening can identify infected decedents whose clinical presentation and terminal event were unrelated to COVID-19, creating a need for integrated interpretation of laboratory, scene, clinical and autopsy information. The present findings suggest the practical value of recording both gross appearances and systematic histology across major organs rather than relying on screening status or pulmonary congestion in isolation. They do not, however, justify treating a test-positive decedent as a COVID-19 death without independent assessment.

Limitations

The principal limitations are the small, single-centre sample; absence of a SARS-CoV-2-negative autopsy comparison group; incomplete details of pre-terminal treatment, clinical severity and post-mortem interval at individual level; and limited contextual evidence for attributing specific lesions to infection. The cohort includes a three-month-old infant, whose normal organ weights differ from adult reference ranges. Some morphological fields were duplicated or overlapping, and formal grading criteria for selected diagnoses were not fully recorded. In addition, the completeness of organ sampling, including brain histology, could not be independently verified from individual tissue-level records. No viral immunohistochemistry or organ-specific tissue RT-PCR results were available to substantiate direct viral involvement. Statistical comparisons were restricted to descriptive patterns because symptom status was associated with the broader natural-versus-unnatural death case mix.

CONCLUSION

Among 30 SARS-CoV-2-positive pre-autopsy screening cases, pulmonary oedema, congestion and haemorrhagic changes were frequently recorded, while histopathological examination identified diffuse alveolar haemorrhage, hyaline membranes, alveolar capillary microthrombi and abnormalities in the heart, liver, kidneys and brain. Findings were present across differing clinical presentations and death circumstances. The results support comprehensive multi-organ documentation during medico-legal autopsy but should not be interpreted as proof that SARS-CoV-2 caused any particular lesion or death. Larger studies with negative controls, standardised pathological criteria and individual clinical correlation are needed.

Declarations

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Conflict of Interest: None declared.

REFERENCES

1. Edler C, Schröder AS, Aepfelbacher M, Fitzek A, Heinemann A, Heinrich F, et al. Dying with SARS-CoV-2 infection-an autopsy study of the first consecutive 80 cases in Hamburg, Germany. Int J Legal Med 2020;134(4):1275-84.
2. Geller RL, Aungst JL, Newton-Levinson A, Smith GP, Mosunjac MB, Mosunjac MI, et al. Is it COVID-19? The value of medicolegal autopsies during the first year of the COVID-19 pandemic. Forensic Sci Int 2022;330:111106.
3. Schaller T, Hirschbühl K, Burkhardt K, Braun G, Trepel M, Märkl B, et al. Postmortem examination of patients with COVID-19. JAMA 2020;323(24):2518-20.
4. Menter T, Haslbauer JD, Nienhold R, Savic S, Hopfer H, Deigendesch N, et al. Postmortem examination of COVID-19 patients reveals diffuse alveolar damage with severe capillary congestion and variegated findings in lungs and other organs suggesting vascular dysfunction. Histopathology 2020;77(2):198-209.
5. Ackermann M, Verleden SE, Kuehnel M, Haverich A, Welte T, Laenger F, et al. Pulmonary vascular endothelialitis, thrombosis, and angiogenesis in Covid-19. N Engl J Med 2020;383(2):120-8.
6. Fox SE, Akmatbekov A, Harbert JL, Li G, Brown JQ, Vander Heide RS. Pulmonary and cardiac pathology in African American patients with COVID-19: an autopsy series from New Orleans. Lancet Respir Med 2020;8(7):681-6.
7. Satturwar S, Fowkes M, Farver C, Wilson AM, Eccher A, Girolami I, et al. Postmortem findings associated with SARS-CoV-2: systematic review and meta-analysis. Am J Surg Pathol 2021;45(5):587-603.
8. Kyada HC, Bhalara RV, Vadgama DK, Varu PR, Trangadia MM, Manvar PJ, et al. Pathological findings in COVID-19: a conventional autopsy-based study from India. Indian J Med Res 2022;155(1):178-88.
9. Ray A, Jain D, Goel A, Agarwal S, Swaroop S, Das P, et al. Clinico-pathological features in fatal COVID-19 infection: a preliminary experience of a tertiary care center in North India using postmortem minimally invasive tissue sampling. Expert Rev Respir Med 2021;15(10):1367-75.
10. Vishwajeet V, Purohit A, Kumar D, Parag V, Tripathi S, Kanchan T, et al. Evaluation of pathological findings of COVID-19 by minimally invasive autopsies: a single tertiary care center experience from India. J Lab Physicians 2021;13(2):97-106.
11. Yadav J, Goel G, Purwar S, Saigal S, Tandon A, Joshi A, et al. Clinical, virological, and pathological profile of patients who died of COVID-19: an autopsy-based study from India. Cureus 2022;14(3):e23538.
12. Santoriello D, Khairallah P, Bomback AS, Xu K, Kudose S, Batal I, et al. Postmortem kidney pathology findings in patients with COVID-19. J Am Soc Nephrol 2020;31(9):2158-67.
13. Halushka MK, Vander Heide RS. Myocarditis is rare in COVID-19 autopsies: cardiovascular findings across 277 postmortem examinations. Cardiovasc Pathol 2021;50:107300.
14. Solomon IH, Normandin E, Bhattacharyya S, Mukerji SS, Keller K, Ali AS, et al. Neuropathological features of Covid-19. N Engl J Med 2020;383(10):989-92.
15. Hammoud H, Bendari A, Bendari T, Bougmiza I. Histopathological findings in COVID-19 cases: a systematic review. Cureus 2022;14(6):e25573.