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Anatomical pathology of covid-19: Postmortem tissue analysis and lessons learned.

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Introduction

Anatomical pathology has played a critical role in elucidating the pathophysiology of COVID-19, especially through postmortem examinations. The analysis of autopsy tissues from individuals who succumbed to SARS-CoV-2 has deepened the understanding of the disease's systemic impact, guiding clinical management, public health responses, and therapeutic strategies.[1].

Early in the pandemic, the lungs were identified as the primary organ affected by COVID-19. Histopathological analyses commonly revealed diffuse alveolar damage (DAD), a hallmark of acute respiratory distress syndrome (ARDS), characterized by hyaline membrane formation, alveolar edema, and type II pneumocyte hyperplasia. Microthrombi in pulmonary vasculature and endothelial damage were also consistent findings, pointing to a significant vascular component in the disease.[2].

Beyond the lungs, systemic involvement became increasingly evident. Cardiac tissues often displayed signs of myocarditis, interstitial infiltration, and microvascular injury, although definitive viral myocarditis remained rare . Renal autopsies frequently showed acute tubular necrosis, glomerular thrombi, and viral particles in renal tissues, suggesting direct and indirect mechanisms of kidney injury [6]. Similarly, hepatic tissues demonstrated moderate steatosis and lobular inflammation, likely due to systemic inflammation, hypoxia, or drug-induced injury [3]

The central nervous system (CNS) findings in COVID-19 autopsies revealed hypoxic-ischemic damage, microglial activation, and inflammatory infiltration, although direct viral invasion was inconsistent . SARS-CoV-2 RNA has been detected in brain tissue, but neuropathological effects are more likely attributable to systemic hypoxia and immune-
mediated damage rather than primary neurotropism
. Lessons learned from postmortem pathology have informed multiple aspects of pandemic response from refining clinical interventions like anticoagulation and steroid use, to supporting vaccine development by highlighting immune responses and tissue damage patterns. Moreover, these insights underline the need for sustained investment in autopsy-based research during infectious disease outbreaks [4].

One of the most significant contributions of anatomical pathology has been the identification of the prothrombotic state in COVID-19. Widespread thrombosis in multiple organs, including the lungs, kidneys, and brain, along with endothelialitis, supports the concept of COVID-19 as a thromboinflammatory disease. These findings influenced the adoption of anticoagulation protocols in hospitalized patients. Limitations of early autopsy studies included biosafety concerns, limited sample sizes, and variations in technique. However, with the establishment of safe autopsy protocols and biobanking initiatives, more comprehensive analyses became feasible. These efforts have revealed that disease manifestations can differ significantly depending on patient age, comorbidities, viral variants, and treatment regimens. [5].

Conclusion

Anatomical pathology, through postmortem tissue analysis, has been indispensable in understanding

COVID-19's multi-organ effects, vascular pathology, and immunologic characteristics. As emerging infectious diseases continue to challenge global health, the role of autopsies remains vital for scientific discovery and clinical preparedness.

References

1. Andreesen R, Brugger W, Löhr GW, et al. Human macrophages can express the Hodgkin's cell-associated antigen Ki-1 (CD30).Am J Clin Pathol. 1989; 134(1):187.
2. Abbondanzo SL, Sato N, Straus SE, et al. Acute infectious mononucleosis: CD30 (Ki-1) antigen expression and histologic correlations.Am J Clin Pathol. 1990; 93(5):698-702.
3. Durkop H, Latza U, Hummel M, et al. Molecular cloning and expression of a new member of the nerve growth factor receptor family that is characteristic for Hodgkin's disease.Cell. 1992; 68(3):421-7.
4. Lakatos PL. Recent trends in the epidemiology of inflammatory bowel diseases: up or down?.World J Gastroenterol. 2006;12:6102-8.
5. Lichtenstein GR, Hanauer SB, Sandborn WJ. Management of Crohn's disease in adults.Am J Gastroenterol. 2009;104:465-83.

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