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# Histopathological Features of Emerging Infectious Diseases: Lessons from COVID-19, Mpox, and Lassa Fever

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**Abstract:** Emerging infectious diseases (EIDs) constitute a persistent and escalating global health challenge. Understanding their histopathological signatures is critical for accurate diagnosis, elucidation of pathogenesis, and guidance of therapeutic strategies. This review examines and compares the histopathological features of three significant EIDs: coronavirus disease 2019 (COVID-19), mpox (formerly monkeypox), and Lassa fever. A narrative literature review was conducted using studies published between January 2000 and December 2024 in PubMed, Scopus, and Web of Science. Keywords were COVID - 19 histopathology, SARS -COV -2 autopsy, mpox pathology, monkeypox histopathology, Lassa fever pathology, and vascular hemorrhagic fever histology. Preference was given to autopsy reports and series of cases of biopsies. Diffuse alveolar damage, microvascular thrombosis, and pulmonary vascular endothelialitis characterize COVID-19. Epidermal ballooning degeneration, intracytoplasmic inclusion bodies, and vesiculopustular eruptions depict Mpox. Lassa fever is characterized by midzonal hepatocellular necrosis, lymphoid depletion, and a surprisingly low extent of inflammatory infiltrate. Each disease shows unique cytopathic effects, inflammatory patterns, and organ tropism that reflect distinct viral pathogenic mechanisms. The histopathological examination cannot be neglected in the study of EIDs. The comparison of the tissue results shows the divergent and convergent pathogenic mechanisms with vital diagnostic and therapeutic implications. The lessons learned during COVID-19, mpox, and Lassa fever outbreaks are cumulative and inform the strategies of readiness in the event of future pandemics.

**Keywords:** Histopathology; emerging infectious disease; COVID -19; SARS-CoV-2; mpox; Lassa fever.

## 1. INTRODUCTION

Emerging infectious diseases (EIDs) are illnesses that have newly appeared in a population or have previously existed but are experiencing a surge in incidence, geographic range or both[1]. It is evident that the world has experienced an EID tsunami over the last thirty years due to ecological disturbance, globalization, climate change, and increasing contact between human, animals and environmental health[2]. Most EID events are caused by viral pathogens, and the causative agent is identified as zoonotic in about 75 percent of the events[3].

In recent years, three viral pathogens have attracted sustained global concern owing to their severe clinical consequences and demonstrated epidemic or pandemic capacity: severe acute respiratory syndrome coronavirus 2, responsible for coronavirus disease 2019; Monkeypox virus, redesignated as mpox by the World Health Organization; and lassa virus, the causative agent of lassa fever. COVID-19 developed into the most consequential pandemic of the present century. Mpox progressed from a geographically restricted zoonotic infection to a multicountry outbreak

recognised as a public health emergency. Lassa fever continues to produce recurrent outbreaks across West Africa and is associated with appreciable case fatality rates[4].

The COVID-19 pandemic generated an unparalleled body of post-mortem material, enabling detailed exploration of the multisystemic nature of SAR-COV-2 infection and the role of immune-mediated tissue injury[5]. The 2022 multicountry mpox outbreak similarly prompted renewed microscopic examination of poxvirus-induced cutaneous pathology. Lassa fever, although previously described in foundational pathological studies, has received renewed scrutiny in light of advances in diagnostic technologies and vaccine research within endemic settings[6].

Although these three diseases have clinical and epidemiological uniqueness, a comparison of the tissue-level manifestations of these conditions has not been done in a systematic manner. The comparison of this type provides an opportunity to identify common pathological pathways, including endothelial dysfunction and immunosuppression, and outline disease-specific features that may serve as diagnostic indicators. This study aims to synthesize the existing knowledge on the histopathology of COVID-19, mpox, and Lassa fever; compare their tissue manifestation at organ systems and draw insights that, perhaps, inform the diagnosis and management of future EIDs.

## 2. METHODOLOGY

The systematic narrative review was carried out in PubMed, Scopus, and Google Scholar databases. Search terms were: COVID-19 histopathology, SARS-CoV-2 autopsy findings, Mpox skin biopsy, Monkeypox histopathology, Lassa fever pathology, and emerging infectious disease histology. Articles published since January 2000 till December 2024 in English and reporting original histopathological data on autopsies, biopsies, or experimental models were included. Additional manual searches of the reference lists of relevant review articles and landmark pathology studies were performed to identify further eligible publications that were not retrieved through the electronic database search. The literature search was conducted independently by the author, and duplicate records were removed before full-text evaluation. Studies were selected based on their relevance to the histopathological manifestations of COVID-19, mpox, and Lassa fever and their contribution to understanding tissue-specific pathological alterations and disease pathogenesis.

## 3. HISTOPATHOLOGICAL FEATURES OF COVID-19

### 3.1 Pulmonary Pathology

The lung is the main target organ of SAR-COV-2, and pulmonary histopathology has been the most widely studied part of COVID-19 pathology. The histopathological correlate of acute respiratory distress syndrome (ARDS), known as diffuse alveolar damage (DAD), is the most common findings in fatal instances of [7]. There are two known stages of DAD, the initial exudative stage and the subsequent organizing/proliferative stage. During the exudative phase, which is usually the first week of severe illness, the alveoli are filled with proteinaceous edema, hyaline membrane formation lines the alveolar walls, and the alveolar capillaries are congested, and type I pneumocytes are lost[8].

The Organizing phase of DAD, which is found in patients who survived beyond 7-10 days, is typified by type II pneumocyte hyperplasia, which is a regenerative response, and by fibroblast proliferation in alveolar spaces, organizing pneumonia pattern, and variable interstitial fibrosis. The presence of multinucleated syncytial giant cells that result from cell fusion caused by the virus has been described as a characteristic though not universal feature of SAR-COV-2 pneumonia[9]. Deposition intra-alveolar fibrin and microthrombi in the capillaries of the alveoli are always reported, which are manifestations of the coagulopathic aspect of severe COVID-19. One of the significant contributions to the understanding of the COVID-19 pneumonia was made by Ackermann in a New England Journal of Medicine article, who found pulmonary vascular endothelialitis to be a characteristic feature of the condition. By comparing COVID-19, influenza, and control lungs, the authors showed intussusceptive angiogenesis a type of vascular remodeling

and viral inclusion structures in endothelial cells on electron microscopy, direct viral infection of endothelium, and significantly increased alveolar capillary microthrombi relative to influenza. Such endothelial tropism makes SARS-CoV-2 unique among other respiratory viral pathogens and the basis of the systemic thrombotic disease[10].

### 3.2 Cardiovascular Pathology

Pathology Histopathology Cardiac involvement in COVID-19 occurs histopathologically as lymphocytic myocarditis, macrophage invasion, myocyte necrosis, and interstitial edema. Myocarditis, though, seems less prevalent than early case reports indicated; a systematic review of COVID-19 autopsy data identified myocarditis in about 7-10% of subjects, and diffuse inflammatory cell infiltration, but not the extent of myocyte necrosis, meeting the Dallas criteria of myocarditis[11]. A pattern of secondary myocardial injury, which can be explained by hypoxemia, cytokine storm, and microvascular thrombosis, but not direct cardiomyocyte infection, is more common[12].

Micro-vascular thrombosis is also a regular observation of the cardiac, pulmonary, and systemic vessels. Myocardium, endothelial swelling, and perivascular inflammation have been reported to contain platelet-fibrin thrombi in the microvasculature, hepatic sinuoids, and cerebral microvasculature[13]. Clinical findings of right ventricular strain are associated with histopathologic findings of right ventricular dilation and microthrombi in pulmonary arterioles.

### 3.3 Renal Pathology

Acute kidney injury (AKI) is a common and prognostically important complication of severe COVID-19. The most common observation by histopathological examination of renal tissue is acute kidney injury (AKI), which is loss of brush border, vacuolization of tubular epithelial cells, nuclear pyknosis, and luminal debris[14]. The systemic prothrombotic state is consistent with peritubular capillary erythrocyte aggregates and glomerular microthrombi. COVID-19 patients, especially those of African descent with high-risk alleles of APOL1 G1 or G2, have been reported to have collapsing glomerulopathy, a severe type of focal segmental glomerulosclerosis, which is characterized by the destruction of the podocyte[15]. Although there are proposals of direct viral infection of tubular epithelial cells based on immune histo-chemical and in situ hybridization studies, the conventional paradigm is that renal injury in COVID-19 is mainly caused by ischemia, cytokine-mediated inflammation and coagulopathy and not direct nephrocytopathic effects.

## 4. HISTOPATHOLOGICAL FEATURES OF MPOX

### 4.1 Skin Pathology

The skill is the main target of mpox. Lesions of mpox typically progress through macular, popular, vesicular, pustular and crusting stages and biopsy specimens of lesions at various stages of development yield correspondingly different histological images[16]. Ballooning degeneration of keratinocytes, reticular degeneration, and single or multiple foci of intraepidermal vesicles are the major epidermal changes observed in vesicular and pustular stages. Ballooning degeneration is the cytoplasmic edema of the infected keratinocytes, which is caused by intracellular edema due to the disruption of cellular homeostasis[17]. Inclusion bodies in the cytoplasm are the most pathogenic characteristic of poxvirus infection in tissues. These eosinophilic intra-cytoplasmic inclusions, which are similar to Guarnieri bodies in smallpox, are viral replication factories called B-type inclusions or Bollinger bodies in mpox[18].

They are the most prevalent in the epidermis and can also be detected in the dermal fibroblasts and macrophages. The inclusions are periodic acid-Schiff (PAS) positive and optimally seen under high magnification on hematoxylin and eosin (H&E) staining. These inclusions are surrounded by a distinct halo in early vesicular lesions following changes caused by the virus in cytoplasmic features[19]. In mpox, the dermis exhibits variable edema, a mixed inflammatory dermal infiltrate, which includes lymphocytes, histiocytes and a few neutrophils, and endothelial swelling with

vascular dilation. As opposed to the vesicles of varicella-zoster virus (VZV) infection, which contain multinucleated giant cells (Tzanck cells) and nuclear inclusions, mpox vesicles are characterized by cytoplasmic inclusions and lack of much multinucleation, a histological difference with diagnostic value[20]. Reticular degeneration and acantholysis may also be seen, and these processes help to create vesicles in the epidermis.

### 4.2 Lymph Node Pathology

The clinically distinctive feature of mpox, which sets it apart of smallpox and varicella, is lymphadenopathy. Affected lymph nodes are histologically characterized by follicular and parafollicular hyperplasia of the lymphocytes, sinuoidal histiocytosis and focal necrosis[21]. In the extreme cases, necrotic foci of lymphoid follicles and subcapsular sinuses could be eminent. Similar inclusion bodies as observed in skin lesions are also observed in lymph node histiocytes. The lymphadenopathy is an expression of a strong local immune response to local viral replication and is a biological indicator of the unique immunopathology of the disease relative to other orthopox viruses.

### 4.3 Visceral Pathology

Systemic mpox spread with visceral involvement, although rare in immune compromised individuals, has been reported in cases of severe infections and in immune deficient individuals. The hepatic involvement appears as focal hepatocellular necrosis with eosinophilic change, and intra-cytoplasmic inclusions in the hepatocytes[22]. In case of pulmonary involvement, interstitial pneumonitis, alongside infiltration of alveolar macrophage and, in the severe cases, DAD-like changes are observed. Spleen can exhibit congestions of white pulp and sinuosity. Gastrointestinal and ulcerative mucosal necrosis has also been reported especially in the 2022 outbreak where proctitis and gastrointestinal manifestations were common in some high-risk groups)[23].

## 5. HISTOPATHOLOGICAL FEATURES OF LASSA FEVER

### 5.1 Hepatic Pathology

The liver is the most common target organ of Lassa virus and hepatic pathology is the most typically described characteristic of the Lassa fever histopathology. The hepatocellular necrosis, with a mainly mid-zonal distribution, is the defining hepatic finding (first described in autopsy series by Walker and colleagues and subsequently confirmed in a number of studies)[24][25]. It is a pattern that occurs in hepatocytes in the mid-portion of the hepatic acinus (Zone 2), but spares the periportal Zone 1 hepatocytes and centrilobular Zone 3 hepatocytes. It is a characteristic that differentiates Lassa fever, which causes mid-zonal necrosis with steatosis, and Ebola and Marburg hemorrhagic fevers, which cause panlobular or Zone 3 predominant necrosis. Lassa fever necrotic hepatocytes exhibit eosinophilic cytoplasmic change, nuclear pyknosis and karyorrhexis, and development of acidophilic Councilman-like (apoptotic) bodies, hyalinized corpses of deceased hepatocytes as in yellow fever[26]. The most important and pathogenic symptom of Lassa fever hepatic pathology is the paucity or even complete absence of inflammatory infiltrate in the presence of a considerable hepatocellular necrosis. This paradoxical dissociation of tissue destruction with inflammation indicates the great immunosuppressive power of Lassa virus, which directly suppresses the activity of dendritic cells, disrupts the signaling of type I interferons, and inhibits the activation of T-cells by binding its surface glycoprotein to alpha-dystroglycan[27].

Additional findings are hepatic sinuoidal congestion with erythrocyte plugging, hyperplasia of Kupffer cells and vacuolar hepatocyte degeneration. Electron microscopy has also described intracytoplasmic viral inclusions in hepatocytes in the form of electron-dense granular structures that are associated with viral ribonucleoproteins. The widespread hepatocyte positivity through the use of immunohistochemical staining of antibodies against Lassa virus antigens shows hepatocytotropism[28].

### 5.2 Splenic and Lymphoid Pathology

There is a significant lymphoid loss in the spleen of fatal Lassa fever that occurs in both white pulp follicles and periarteriolar lymphoid sheaths (PALS). This observation is similar to the lymphoid depletion observed in other viral hemorrhagic fevers, including Ebola and Marburg, and is in line with the severe lymphopenia that is clinically apparent[29]. Focal necrosis of parenchyma of the spleen, sinusoidal congestion, and hemophagocytosis, ingestion of erythrocytes and of leukocytes by activated macrophages, may be present. Lymphoid depletion is an expression of direct viral-induced lymphocyte apoptosis, dysfunctional lymphocyte trafficking and immune fatigue (mediated by cytokines)[30].

The histology of lymph nodes is similar to that of the spleen, and includes follicular depletion, paracortical atrophy, and sinus histiocytosis. The virtual lack of follicular germinal centers is associated with the inability to develop an efficient humoral immune response, which is one of the factors of high case fatality and the inability to produce a protective immunity in the long term[31].

### 5.3 Adrenal, Pulmonary, and Other Organ Pathology

Involvement of adrenal glands in Lassa fever involves focal cortical necrosis, hemorrhage and inflammatory infiltration, which can lead to the vascular collapse and shock observed in severe cases[32]. The adrenal cortex pathological changes might disrupt the production of glucocorticoids, which may further deteriorate the hemodynamic instability. Pulmonary histopathology shows interstitial pneumonitis, accumulation of alveolar macrophages, interstitial edema and fibrin deposition. These pulmonary changes, which are different than the DAD of COVID-19, indicate hematogenous seeding of lung parenchyma with Lassa virus and vascular permeability mediated by cytokines[33]. The histopathological changes in the kidney in Lassa fever comprise glomerular fibrin thrombi, mesangial proliferation, and tubular necrosis, which are in line with the acute kidney damage, as seen in the clinical scenario. Immune complex deposition of glomeruli has been reported but the level of immune complex-induced glomerulonephritis compared to direct viral damage and ischemic damage needs to be demystified. Hemorrhagic presentations of Lassa fever seen in some 20-30 percent of hospitalized patients associated with extensive endothelial dysfunction, platelet dysfunction and inhibited coagulation instead of the vasculitic endothelial necrosis of Ebola virus disease.

## RESULTS AND DISCUSSION

| Parameter | COVID-19 | Mpox | Lassa Fever |
|---|---|---|---|
| Inflammatory infiltrate | Variable - cytokine storm pattern; lymphocytic/macrophage infiltration | Mixed - lymphocytes, histiocytes, neutrophils in Dermis | Paradoxically minimal despite necrosis |
| Lymphoid pathology | Lymphocytic depletion in severe disease | Follicular hyperplasia; lymphadenopathy distinctive | Severe lymphoid depletion, splenic white pulp atrophy |
| Hepatic findings | Microvascular steatosis, lobular inflammation, pericentral necrosis | Focal hepatocyte necrosis with inclusions (rare visceral spread) | Mid-zonal necrosis, Councilman bodies, Kupffer cell hyperplasia |
| Hemorrhagic | Microthrombi rather than | Uncommon in typical | Present in ~20–30%; |

| | | | |
|---|---|---|---|
| features | true hemorrhage | disease; necrotic pustules in severe cases | coagulopathy-mediated, not vasculitis |
| Best diagnostic tissue | Lung (autopsy/BAL), heart, kidney | Skin biopsy (vesicular/pustular stage) | Liver biopsy (postmortem); blood for RT-PCR preferred antemortem |
| Special stains/IHC | Anti-SARS-CoV-2 IHC, CD61 (platelets), CD3/CD68 | H&E sufficient; PAS/IHC for confirmation | Anti-Lassa IHC, EM for inclusions |
| Immune evasion mechanism | Cytokine dysregulation; type I IFN suppression | Viral immunomodulatory proteins; less pronounced | Profound IFN suppression; DC dysfunction; T-cell impairment |
| Primary target organ | Lungs | Skin | Liver |
| Key hallmark lesion | Diffuse alveolar damage (DAD) with hyaline membranes | Epidermal ballooning degeneration + intracytoplasmic inclusions | Mid-zonal hepatocellular necrosis with minimal inflammation |
| Inclusion bodies | Absent (viral particles by EM only) | Present - intracytoplasmic (B-type/Bollinger bodies) | Intracytoplasmic (by EM); not routinely visible on H&E |
| Vascular involvement | Severe - endothelialitis, microthrombi, intussusceptive angiogenesis | Mild - dermal vascular dilation, endothelial swelling | Moderate - endothelial dysfunction, glomerular thrombi |

Lassa fever across organ systems and diagnostic parameters (Table 1)

Table 1. Comparative histopathological features of COVID-19, mpox, and Lassa fever. BAL = bronchoalveolar lavage; DAD = diffuse alveolar damage; DC = dendritic cell; EM = electron microscopy; H&E = hematoxylin anaeosin; IFN = interferon; IHC = immune-histochemistry; PAS = periodic acid-Schiff.

## 6. COMPARATIVE HISTOPATHOLOGICAL ANALYSIS

### 6.1 Comparative Table

The following table summarizes the key histopathological features of COVID-19, mpox, and Lassa fever across organ systems and diagnostic parameters (Table 1).

### 6.2 Convergent and Divergent Pathogenic Mechanisms

Although the clinical manifestations of COVID-19, mpox, and Lassa fever are vastly different, the three pathogens are convergent at the histopathological level. A commonality between the three diseases is endothelial dysfunction. In COVID-19, SARS -COV -2 directly infects endothelia through the ACE2 receptor, resulting in endothelialitis and severe procoagulant state[34]. The endothelialitis dysfunction in Lassa fever is an indirect process involving cytokine-mediated vascular permeability, platelet aggregation inhibition, and consumption of coagulation

factors without the overt vasculitis observed in filoviral hemorrhagic fevers. Mpox has shown to exhibit relatively mild endothelial effects, which is mainly dermal vascular dilation and edema, as opposed to systemic thrombotic microangiopathy.

Another convergent feature is lymphoid depletion. Each of the three pathogens disrupts adaptive immunity by depleting lymphoid populations either directly by causing cytopathic defects of viruses or by promoting apoptosis or indirectly by cytokine-mediated immune exhaustion. Lassa fever and fulminant COVID-19 have the most significant lymphoid depletion and show near-complete atrophy of the spleen white pulp, which is a histopathologic finding consistent with the immune evasion mechanisms of these two pathogens[35]. The conflicting sides are also educative. Histopathological diagnostic markers of poxvirus infection are easily accessible by intracytoplasmic inclusion body in mpox that is absent in COVID-19 and Lassa fever on routine light microscopy. The hepatic necrosis occurs in a mid-zonal pattern in Lassa fever which is quite specific enough especially when it is accompanied by paucity of inflammation to provoke high suspicion of arenavirus infection despite no molecular validation. On the other hand, the vascular endothelial-damage-associated diffuse alveolar damage in COVID-19 has no analogous similarity in the other two diseases and indicates the specific pulmonary tropism and thrombo-inflammatory physiology of SARS-CoV-2.

## 7. LESSON LEARNED AND FUTURE DIRECTIONS

### 7.1 Histopathology as an Early Warning and Characterization Tool

Among the most valuable lessons of COVID-19 pandemic is the importance of systematic autopsy tissue analysis at the beginning of an outbreak. Within the first weeks of the COVID-19 pandemic, the results of autopsy in China and Europe quickly defined DAD as the primary pulmonary pathology, the procoagulant vascular phenotype, and multiorgan involvement, which were directly applied to clinical management guidelines and the design of therapeutic trials. The speed with which these findings were published by preprint servers, despite concerns over quality control, increased the pace of the worldwide scientific response and provided evidence of how organized autopsy networks can be used in outbreak situations. In the case of Lassa fever and mpox, which have been known to have epidemic potential since decades, the available histopathological literature was a basis on which the clinical teams could rely on in the face of mounting outbreaks. Nevertheless, there are still important gaps: the histopathology of mpox among immune compromised patients, the tissue-level correlates of post-acute sequelae of COVID-19 (long COVID) and the histopathology of Lassa fever in children are some areas that still need research[36] .

### 7.2 Implications for Pandemic Preparedness

A comparative analysis of the histopathology of these three EIDs brings about a number of lessons that have direct implication in the preparedness against pandemics. First, the key role of endothelial dysfunction in all three diseases supports the idea of tracking vascular biomarkers, such as endothelin-1, von Willebrand factor, and angiopoietin-2 as possible markers of disease severity and interventions to mitigate it. Second, the paradoxical dissociation between tissue damage and inflammation in Lassa fever and to a lesser extent in COVID-19 cytokine storm, shows that clinical indicators of inflammation do not necessarily correspond to the extent of tissue damage, and applies to risk stratification algorithms. Third, diagnostic utility of skin biopsy in mpox emphasizes the role of tissue-based diagnostics in case of molecular tests. PCR resources are scarce in outbreak contexts; trained pathologists with the capacity to identify intracytoplasmic inclusions and epidermal ballooning degeneration could be used to give quick presumptive diagnoses, which would be used to justify sufficient isolation and contact tracing. Pathology infrastructure, especially in West Africa where Lassa fever is endemic, and countries with limited diagnostic potential, is an essential part of EID preparedness .

### 7.3 Long COVID and Post-Infectious Pathology

The phenomenon of post-acute sequelae of SARS-CoV-2 infection (PASC), or long COVID, has emerged as a major public health challenge affecting an estimated 10–30% of COVID-19 survivors. Histopathological investigation of long COVID patients through biopsy studies has revealed persistent microglial activation in the brain, ongoing endothelial dysfunction, fibrotic remodeling of pulmonary parenchyma, and evidence of viral RNA persistence in multiple organs including the gut mucosa[37]. These findings challenge the assumption that viral clearance equates to complete tissue recovery and suggest that EID management must include long-term tissue-level surveillance for evidence of persistent pathology.

The post-infectious sequelae of mpox, including scarring, post-inflammatory hyperpigmentation, and rare cases of corneal scarring and orchitis, have counterparts in the histopathological picture of the healing phase of mpox lesions, wherein fibrotic repair tissue replaces necrotic epidermis[38]. Lassa fever survivors may experience sensorineural hearing loss, a complication affecting approximately 30% of patients, and preliminary evidence suggests that immune complex deposition in cochlear tissue may play a role, though definitive histopathological studies in human cochlear tissue remain sparse[39].

## CONCLUSION

This review has shown that COVID-19, mpox, and Lassa fever have unique and diagnostically informative histopathological phenotypes that capture their unique viral biology, tropism and immunopathogenesis. COVID-19 diffuse alveolar damage and vascular endothelialitis, the epidermal ballooning degeneration and intracytoplasmic inclusions of mpox, and the mid-zonal hepatocellular necrosis with paradoxically paucicellular inflammation of Lassa fever are all histopathological fingerprints that, when identified by trained pathologists, allow diagnosis and clinical management.

Convergent themes, such as endothelial dysfunction, lymphoid depletion, and immune evasion, reflect common pathogenic susceptibilities that can be addressed by general-purpose therapeutic interventions, such as anticoagulants, immunomodulators, and endothelial protective reagents. Disease specific diagnostic anchors that are of specific value in resource constrained environments are divergent features, especially the intracytoplasmic inclusions found in mpox and the zonal hepatic necrosis of Lassa fever. With the world community struggling against an increasing trend of EID outbreaks, the need to invest in autopsy programs, histopathology infrastructure, biosafety training and the digital pathology platform is not just a scholarly endeavor but a social health requirement. The experience encoded in the tissue sections of the deceased victims of COVID-19, mpox, and Lassa fever continues to pass on essential information that would save lives during the next outbreak. The science of disease, known as histopathology, is as topical as ever at the interface of health security in the world.

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