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# Full autopsy in a confirmed COVID-19 patient in Lagos, Nigeria – A case report

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ABSTRACT

*Objectives:* To report the postmortem findings of a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) positive individual who died in Lagos (Nigeria) in June 2020 and to investigate the cause, pathogenesis as well as pathological changes noticed during the examination.
*Methods:* Complete postmortem examination was performed according to standard procedures in a regular autopsy suite using personal protective equipment including N95 masks, goggles and disposable gowns. The diagnosis of coronavirus disease 2019 (COVID-19) was confirmed by real-time reverse transcription polymerase chain reaction (RT-PCR) testing on postmortem nasopharyngeal swabs.
*Results:* A 47-year-old man with a medical history of well controlled hypertension and dyslipidaemia died after long hours of transportation for medical care in a hospital in Lagos. He tested positive for SARS-CoV-2 on ante- and postmortem nasopharyngeal swabs. Autopsy revealed pneumonia with diffuse alveolar damage, disseminated intravascular coagulopathy and hypovolaemic shock.
*Conclusions:* Autopsy can be performed on decedents who died from or with SARS-CoV-2 infection in a low resource environment such as ours. A standard autopsy room was used while deploying recommended infection prevention control and regular decontamination. The clinical details, autopsy findings such as diffuse alveolar damage and airway inflammation were consistent with a COVID-19 related pathology. While the decedent had 'controlled' co-morbidity, he succumbed to multi-organ failure occasioned by shock and disseminated intravascular coagulopathy.

## 1. Introduction

Coronavirus Disease 2019 (COVID 19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). This enveloped non-segmented positive sense single-stranded RNA virus is the cause of the global pandemic causing up to 156.5 million infections and 3.3 million deaths worldwide as at 8th of May 2021. Africa has had 3.4 million infections and 83,785 deaths, while Nigeria reported 165,352 cases and 2,065 deaths. [1]

There are several articles on the pathologic manifestations of COVID 19. Barton et al reported 2 cases in which full autopsies were performed in Oklahoma, USA.[2], While some have studied autopsy findings in decedents who succumbed to the virus in the community, others have simply focused on pathologic findings from the lungs of decedents who were treated for the infection and those who had lobectomy for cancer and later found out to be COVID-19 positive. [3–8] Large multicentric study by Hooper et al summarised finding from survey of 135 autopsies in a multi-institutional setting and detailed the frequency of co-morbidities in the subjects. [9]

COVID-19 joins severe acute respiratory syndrome coronavirus

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(SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV) in causing significant morbidity and mortality. [10] Most individuals are asymptomatic or express only mild symptoms, however, some individuals deteriorate rapidly and develop acute respiratory distress syndrome (ARDS). [2,11]

The reported low incidence and death rate in Africa coupled with the lack of biosafety level 3 (BSL-3) autopsy facility may perhaps be responsible for the dearth of articles on COVID-19 related deaths and autopsies. We are aware of few publications in this regard as at the time of putting together this manuscript.

The aim of this article is to report the postmortem findings of a severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) positive individual who died in Lagos, Nigeria in June 2020 and to investigate the cause, pathogenesis as well as pathological changes noticed during the examination.

## 2. Materials and Methods

The venue was a privately managed government-approved multipurpose mortuary facility for body storage, embalmment and autopsies. The facility was situated on an acre of land with adjoining reception, halls for funeral rites, autopsy suite, changing room, offices and a cremation centre. The autopsy suite was well lit with 4 regular dissecting tables, good hygiene with 2 extractor fans and functioning air conditioners.

The procedure was performed by 5 personnel with the following assigned job roles;

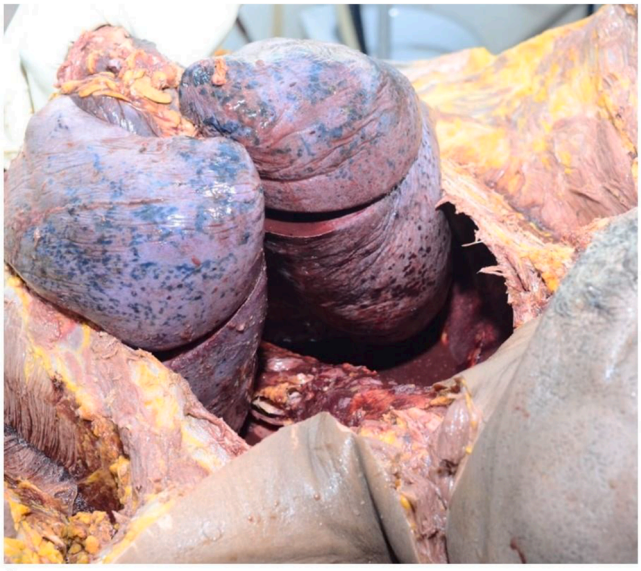
1. Main Pathologist – directed the proceedings, evisceration and organ dissection
2. Assistant Pathologist - evisceration and organ dissection
3. Mortician – Removal of calvarium, instrument handling, cleaning and body reconstruction
4. Photographer – autopsy photos
5. Safety officer – intermittent spraying of all personnel with hypochlorite and general note taking.

Consent for autopsy was obtained from the decedent's sister who was the next of kin.

Swab samples were taken from the nasopharynx during external examination while that of the lungs were taken in a sterile manner after an incision of the parenchyma. The swabs were immediately placed in different viral transport media (VTM) and sent to the Lagos State Biobank for real-time reverse transcription polymerase chain reaction testing for SARS-CoV-2. The vitreous humour and urine were also sampled for SARS-CoV-2 testing.

Representative sections of tissues were fixed in 10% formol saline, processed, embedded in paraffin, cut onto glass slides and stained with haematoxylin and eosin (H&E) in the usual fashion. Immunohistochemistry was performed using appropriate positive and negative controls for CD68, CD5 and CD20 to better characterize the inflammatory cell infiltrates. All slides from the organs were examined by two (2) histopathologists who are fellows of the National Postgraduate Medical College of Nigeria with over 10 years of experience. The formalin fixed and paraffin embedded tissue blocks and stained slides were retained indefinitely.

The body was already embalmed to possibly reduce virulence or level of infectiousness of SARS-CoV-2. A pre-autopsy meeting was conducted in which specific roles were assigned including emergency protocols in case of accidents. Extra precautions were taken by wearing full body impervious suits, face shields, N95 and surgical masks, boots, elbow length gloves (doubled) and use of hypochlorite as well as alcohol-based solution for intermittent hand cleansing and general disinfection during the autopsy. [12–13]



A

B

**Fig. 1.** Photo of right haemothorax with haemorrhagic pleuritis (A) and generalised congestion of the left lung cut surfaces (B).

## 3. Case report

The decedent was a 47-year-old man who worked as an administrator for an oil servicing company, with a medical history of well controlled hypertension and dyslipidaemia. He had previously been quarantined for 16 days in a holding facility onshore and certified fit for an offshore assignment. Standard procedure adopted by the company at that time was to quarantine staff scheduled for offshore duties in a holding facility and also examine for fitness before embarking on the assignment.

He was well until about 29th of May 2020 while on board a vessel in transit to an offshore platform. He developed malaria-like symptoms and was treated as such but his illness deteriorated into a respiratory condition necessitating evacuation on the night of 3rd of June 2020 from the offshore platform. He arrived an onshore facility around 12p.m. the next day and was moved via an ambulance to a hospital in Lagos around 3:45p.m. but was not admitted because of administrative challenges until 7:57p.m. in another medical facility. Hospital notes showed that he was breathless as at the time of admission and eventually became

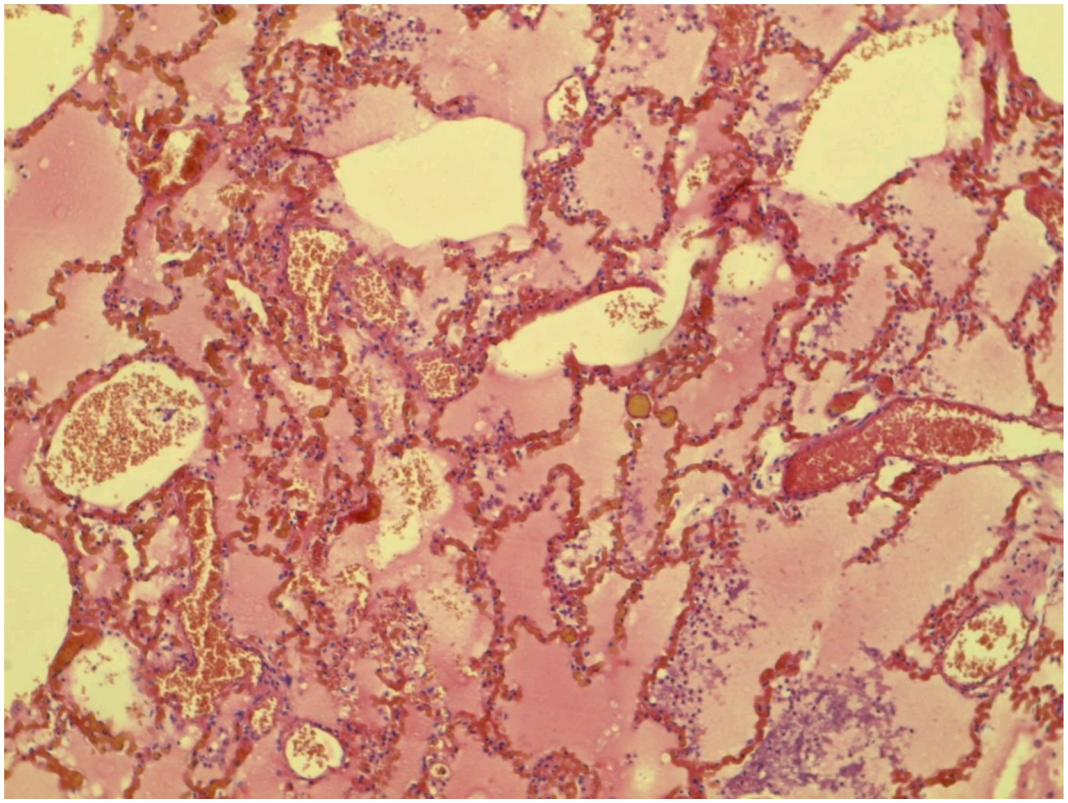


**Fig. 2.** Photomicrograph showing interstitial and intraluminal inflammatory cell infiltrates, congested blood vessels with DAD, H&E x100.

unconscious around 10:36p.m. with history of serial episodes of low blood pressure.

He was managed as a case of COVID-19 related illness with hypovolaemic shock till he died on the 5th of June around 2:30p.m. Naso- and oropharyngeal swabs taken for COVID-19 test before he died were positive.

## 4. Results

The decedent was a well-built average height (172.0 cm in length) negroid male with altered blood-filled nasal cavities. The internal examination revealed focal haemorrhagic adhesions at the base with a pericardium enclosing an enlarged heart that weighed 550 g (300–350 g). There was obvious left ventricular free wall hypertrophy measuring 2.30 cm (1.00–1.50 cm). [14]

The chest cavity showed haemothorax containing 700 mL of blood with extensive haemorrhagic pleuritis and generalised congestion on cut surfaces (Fig. 1). The lungs were heavy weighing 850 g (325–580 g) each. [15] Histologic sections of the lungs showed diffuse alveolar wall damage with fibrin filled spaces, haemorrhagic inspissation, mixed inflammatory cell infiltration (Fig. 2) and focal microthrombi.

A large number of CD68-positive macrophages were mainly localized around the alveolar lumina and septae while other areas showed sparse infiltrates of CD5-positive T-lymphocytes and rare CD20-positive B-lymphocytes (Fig. 3).

There was evidence of massive upper gastrointestinal haemorrhage with inflamed mucosal lining - the oesophagus and stomach contained 200 mL and 1000 mL of altered blood respectively. Histologic sections of the stomach showed partial autolytic changes with areas of ulceration, oedema, thrombi (Fig. 4) and congested blood vessels.

The overall picture of the kidneys showed acute tubular necrosis with prominent corticomedullary differentiation on cut sections and ghost appearance of epithelial cells of most of the proximal tubules with indiscernible nuclear outlines on histology.

The prostate gland was enlarged weighing 150 g [14] with histologic sections that displayed proliferated and variably infarcted glands within an abundant fibromuscular stroma. Also noticed were focal areas of microthrombi. The spleen was slightly enlarged 200 g (45 – 190 g) [16] with evidence of splenitis on histologic examination.

The postmortem nasopharyngeal swab was positive for SARS-CoV-2 while that of the lung parenchyma, urine and vitreous humour were negative.

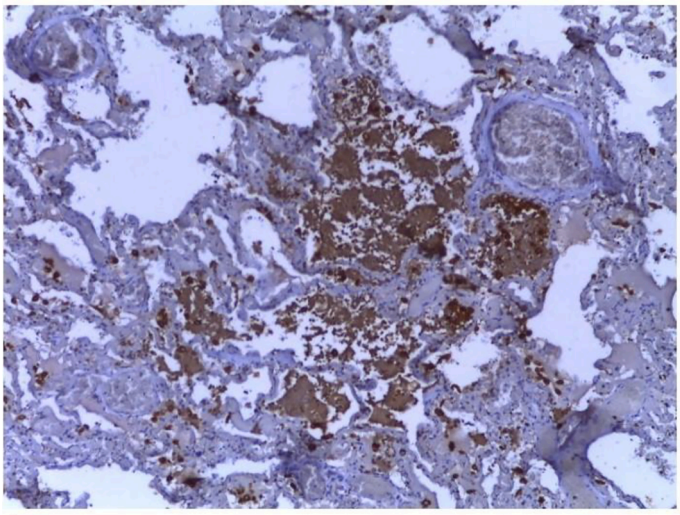
In the final autopsy report the cause of death was reported as haemorrhagic (hypovolaemic) shock due to Disseminated Intravascular Coagulopathy from COVID-19 infection. Hypertensive Heart Disease was listed as a contributory factor.

## 5. Discussion

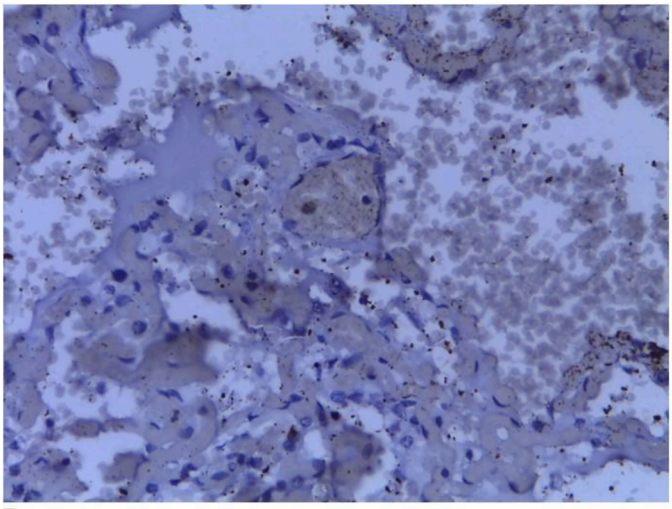
To the best our knowledge this is the first reported case of a full autopsy performed on the body of a SARS-CoV-2 infected person in Nigeria.

Virtually all decedents cited in the autopsy survey study by Hooper et al were reported as having more than one pre-existing condition - a combination of prior chronic disease and acute conditions acquired during hospitalization. [9] The deceased in this report was reported to be hypertensive albeit controlled.

The SARS and MERS-related coronavirus are both considered hazard group 3 (HG3) pathogens. HG3 organisms require risk assessment, previous understanding of the pathology and universal standard precautions and procedures for specific HG3 pathogens. [17] Postmortems should be undertaken in specific premises with adequate ventilation or down-drafts at the workstations and performed under well established specifications and special personal protective equipment. This is part of the Royal College of Pathologists' guidelines with the summary and interpretation about how to perform autopsies on bodies suspected to have died of/with COVID-19. [18] We practice in a low-resource environment with unique problems necessitating performance in a less than



A



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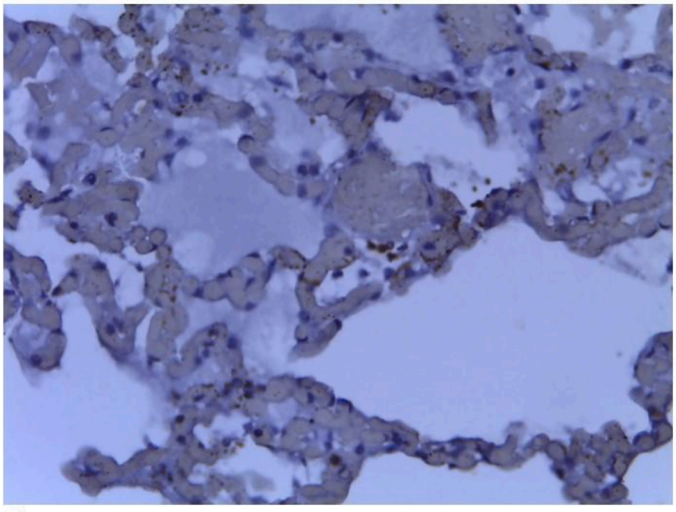

C

**Fig. 3.** Photomicrographs of immunohistochemical stains showing prominent CD68-positive macrophages, x40 (A), sparse CD5-positive lymphocytes, x100 (B) and rare CD20-positive lymphocytes, x400 (C).

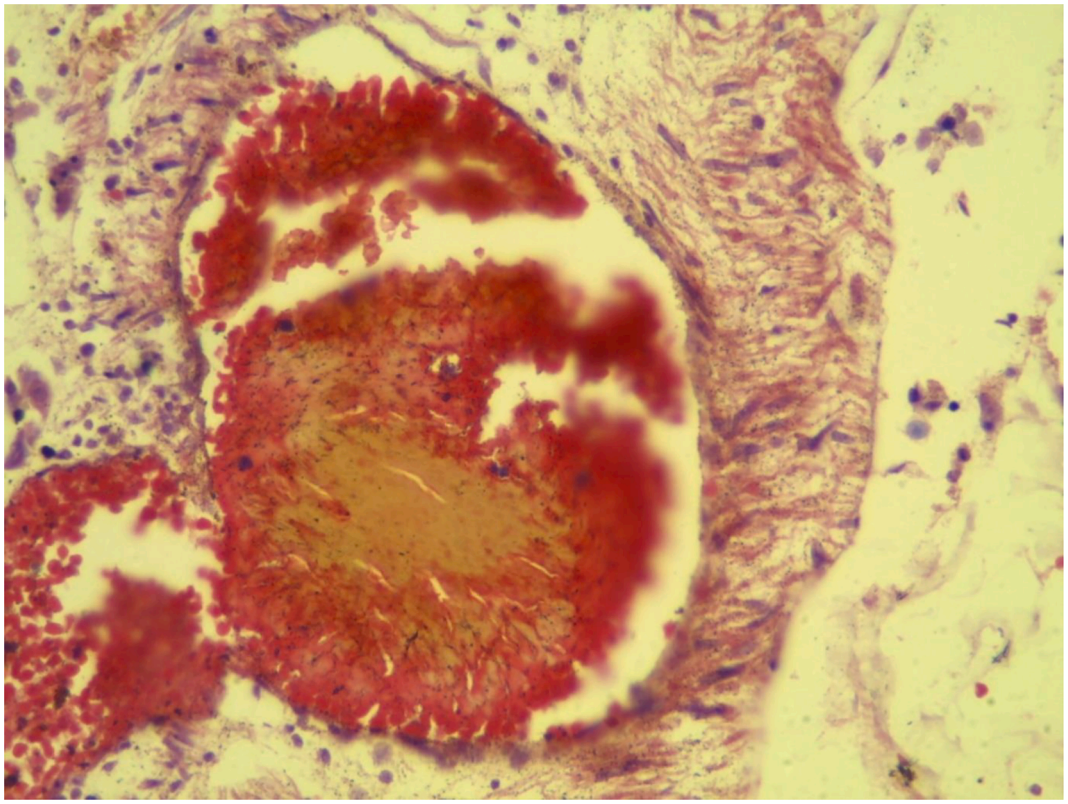

**Fig. 4.** Photomicrograph showing gastric mucosa with a thrombus, H&E x400.

ideal condition. Necessary precaution was taken to minimise the risk of infection during the autopsy.

Our findings of diffuse alveolar wall damage (DAD) with fibrin filled spaces, haemorrhage, microthrombi, inflammatory exudate and congested vessels in the lungs agree with the work of Carsana et al who reported these features as part of the main histological findings from the lung tissues of 38 cases who died from COVID-19. [19] Despite the importance of lung involvement in COVID-19 patients, only limited data are available concerning lung pathology. A paper from China described the histological lesions in a patient who died of COVID-19 - desquamation of pneumocytes, DAD and oedema similar to what was noticed in this case. [20] Other authors described pulmonary pathology of early phase COVID-19 in two patients with lung carcinoma; both patients exhibited signs of the exudative phase of DAD. [8] The index case showed features of acute exudative phase of DAD. Autopsy studies of deceased patients with severe COVID-19 often described the presence of DAD. However, there is an ongoing discussion about the specificity of these changes as DAD seen in other viral infections show similar features. [7]

Immunohistochemistry confirmed the presence of large number of macrophages (CD68-positive macrophages) and this supports the findings of Carsana et al where a large number of CD68-positive macrophages were mainly localized in the alveolar lumens. [19] As detailed by Hanley et al in a postmortem study describing the histopathological findings and viral tropism in UK patients, the inflammation in the lung comprised a predominance of interstitial macrophages along with scattered plasma cells and mild to moderate CD4-positive T-cell lymphocytic inflammation. [21]

Clinical studies have shown that COVID-19 is complicated or related to coagulopathy and thrombosis. Small case reports and case series have demonstrated the presence of fibrinous exudates and microthrombi in histopathological examinations in patients with COVID-19. [22] We were able to demonstrate the presence of microthrombi in the vessels of the lungs, stomach and prostate. Carsana et al in their study observed fibrin thrombi in 33 out of 38 patients, half of them with greater than 25% of tissue involvement and associated with high levels of D-dimer. [19] D-dimer was not assayed in this patient.

In addition to the thrombotic complications, other extrapulmonary manifestations include myocardial dysfunction and arrhythmia, acute coronary syndromes, acute kidney injury (AKI), gastrointestinal symptoms, hepatocellular injury, hyperglycemia and ketosis, neurologic illnesses, ocular symptoms and dermatologic complications. This pathology may reflect either extrapulmonary dissemination and replication of SARS-CoV-2 as has been observed for other zoonotic coronaviruses [23,24] or widespread immunopathological sequelae of the disease. Key mechanisms that may have a role in the pathophysiology of multi-organ injury secondary to infection with SARS-CoV-2 include direct viral toxicity, endothelial cell damage and thromboinflammation, dysregulation of the immune response, and dysregulation of the renin–angiotensin–aldosterone system (RAAS). There is an emerging conversation around myocardial injury in COVID-19 patients, and many in the medical community are wondering whether tissue examination will reveal evidence of myocarditis in these patients. [2,24] In the Hooper et al series myocarditis was rarely cited. [9] The index patient had evidence of hypertensive cardiovascular disease just like previously cited case reports. [2] We did not observe any evidence of myocarditis.

Acute kidney injury is a frequent complication of COVID-19 and is associated with mortality. [25] AKI occurred at much higher rates in critically ill patients admitted to New York City hospitals, ranging from 78% to 90%. [25] Several possible mechanisms specific to SARS-CoV-2 that distinguish this renal abnormality from the more general AKI that accompanies severe illness are noteworthy. First, SARS-CoV-2 may directly infect renal cells, a possibility supported by histopathology findings and the presence of ACE2 receptors. [26] Others are the demonstration of lymphocytic endothelialitis in the kidney and cytokine storm. [27,28] Individuals who succumbed to AKI from direct viral infection presented with histopathological findings which included

prominent acute tubular injury, diffuse erythrocyte aggregation, and obstruction in peritubular and glomerular capillary loops, in addition to the demonstration of viral inclusion particles by electron microscopy. [26–29] It appears the index patient had renal features in keeping with evidence of hypovolaemic shock in the form of ghost appearance of epithelial cells of most of the proximal tubules with indiscernible nuclear outlines. This is not unexpected as other potential aetiologies of AKI common to critical illness presentations, including ARDS, rhabdomyolysis, volume depletion, and interstitial nephritis have been documented and remain relevant in patients with COVID-19. [30]

Evidence of upper gastro-intestinal bleeding was documented in the decedent. The pathophysiology of gastrointestinal tract (GIT) damage in COVID-19 is multifactorial and includes virus-mediated direct tissue damage, microvascular small-bowel injury and alteration of the intestinal flora by the virus. [27,31,32] However, no GIT symptom was reported antemortem hence the obvious conclusion will be a DIC occurring about the time of death.

The detection of viral antigen positivity in the nasopharyngeal swab one week after death and despite full embalmment emphasises the need for caution during autopsy. Whilst a positive RT-PCR test may not necessarily equate infectivity, the risk of infection in non-embalmed or freshly embalmed body becomes high during evisceration and delivery of the tongue and pharyngeal tissue which may cause splattering and dissemination of aerosolized particles. Adequate use of complete PPE will reduce the risk of transmission at autopsy. We also suggest the administration of formalin-soaked gauze through the nostrils to the pharynx to incapacitate the virus before autopsy. If available, autopsy should be done in a dedicated Biosafety Level 3 (BSL 3) facility.

## 6. Conclusion

We present the autopsy findings in a SARS-CoV-2 positive patient who died from life-threatening pulmonary complications with DIC and hypovolaemic shock. While the decedent had ‘controlled’ co-morbidity, he succumbed to multi-organ failure. A standard autopsy room was used while employing improvised infection prevention strategy. Autopsy findings such as diffuse alveolar damage and airway inflammation are consistent with true virus-related pathology.

## 7. Ethics statement

The case report in this paper adhered to the Declaration of Helsinki guidelines.

A written and informed consent was obtained from the decedent’s next of kin (sister) for the postmortem procedure.

The Ethics Committee of the Lagos University Teaching Hospital (LUTH), Idi-Araba, Lagos, Nigeria also granted the authors the consent for publication of this paper.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Mr Johnny Oveghor – Photographer.
Dr Kingsley Obule – Logistics.
Dr Olakunle Odeyemi – Logistics.
Mr Abdulateef Olanlege – Logistics.
Family of the decedent – Consent for autopsy.

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Y. Liu, Y.-H. Hu, P. Peng, J.-M. Wang, J.-Y. Liu, Z. Chen, G. Li, Z.-J. Zheng, S.-Q. Qiu, J. Luo, C.-J. Ye, S.-Y. Zhu, N.-S. Zhong, Clinical Characteristics of Coronavirus Disease 2019 in China, N Engl J Med. 382 (18) (2020) 1708–1720, https://doi.org/10.1056/NEJMoa2002032.

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