Forensic Science, Medicine and Pathology (2022) 18:369–381
https://doi.org/10.1007/s12024-022-00494-1

REVIEW

Findings and inferences from full autopsies, minimally invasive autopsies and biopsy studies in patients who died as a result of COVID19 — A systematic review

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Accepted: 7 June 2022 / Published online: 8 July 2022

Abstract
Many articles on COVID19 deaths have been published since the pandemic has occurred. On reviewing the articles published until June 2021, the findings were very heterogeneous. Adding to the existing knowledge, there were also some unique observations made in the pathogenesis of COVID19. This review was done to determine the findings obtained and inferences drawn from various studies published globally among patients who died due to COVID19. PRISMA guidelines were used to conduct this systematic review. A search of databases like PubMed, ScienceDirect and Epistemonikos was done. The articles focusing on postmortem sample studies involving full autopsies, minimally invasive autopsies and tissue biopsy studies were screened and searched. The studies included were all the case reports, case series, narrative reviews and systematic reviews obtained in full text and in the English language containing study information, and samples obtained postmortem. The information obtained was tabulated using Microsoft excel sheets. The duplicates were removed at the beginning of the tabulation. Zotero referencing software was used for article sorting and citation and bibliography. Two authors independently reviewed the articles throughout the process to prevent bias. Adding to the heterogeneity of COVID19, the concept of lethality in preexisting disease conditions, the occurrence of secondary bacterial and fungal infections, and other pathogenetic mechanisms uniquely encountered are to be considered in treating the patients. Also, the presence of SARS-CoV-2 postmortem is established and should be considered a hazard.

Keywords Autopsy findings in SARS-CoV-2 deaths · Forensic autopsy in COVID19 deaths · COVID19 histopathology findings · Autopsy and COVID19 deaths

Introduction

The WHO Coronavirus (COVID19) live dashboard on 08 September 2021 displayed 221,134,742 confirmed cases including 4,574,089 deaths globally. The WHO weekly situation report displays that the global incidence of COVID19 cases remained stable over the month with over 4.4 million new cases reported between 30 August 2021 and 05 September 2021, with just 64,000 deaths. All regions reported a declining trend of a similar trend except in regions of the Americas where there was a 19% increase comparatively [1]. Many articles have been published since the pandemic has occurred. Though there was hesitancy in conduction of postmortem/autopsy examination on COVID19 deaths, many autopsy surgeons/pathologists have conducted autopsies to reveal the heterogeneity of COVID19. Upon reviewing the articles published until June 2021, the findings were more in the lungs, heart and coagulopathy in general. But adding to the existing knowledge there were also some unique observations made in the pathogenesis of COVID19.

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Rationale and objective of the review This review was done to determine the findings obtained and inferences drawn from various studies published globally among patients who died due to COVID19.

Methods The current review was done according to Preferred Reporting Items for Systematic Review (PRISMA) guidelines [2]. A thorough search of databases including PubMed, ScienceDirect and Epistemonikos was done for the articles focusing on postmortem sample studies involving full autopsies, minimally invasive autopsies and tissue biopsy studies. The search terms used were "autopsy findings in SARS-CoV-2 deaths", "forensic autopsy in COVID19 deaths", "COVID19 histopathology findings" and "autopsy and COVID19 deaths". The cross-references were done accordingly. The studies included were all the case reports, case series, narrative reviews, and systematic reviews obtained in full text and in the English language containing information on study samples obtained post-mortem. Few comparative studies were also selected. The articles with live sample study, without complete articles, and from other languages were excluded. Month /year wise studies with the author details, findings obtained, and the inferences drawn were tabulated using Microsoft Excel sheets. The duplicates were removed at the beginning of the tabulation. Zotero referencing software was used for article sorting and citation and bibliography. Two authors independently reviewed the articles throughout the process to prevent bias. (please see Fig. 1).

The findings of the study are summarized below (please see Table 1).

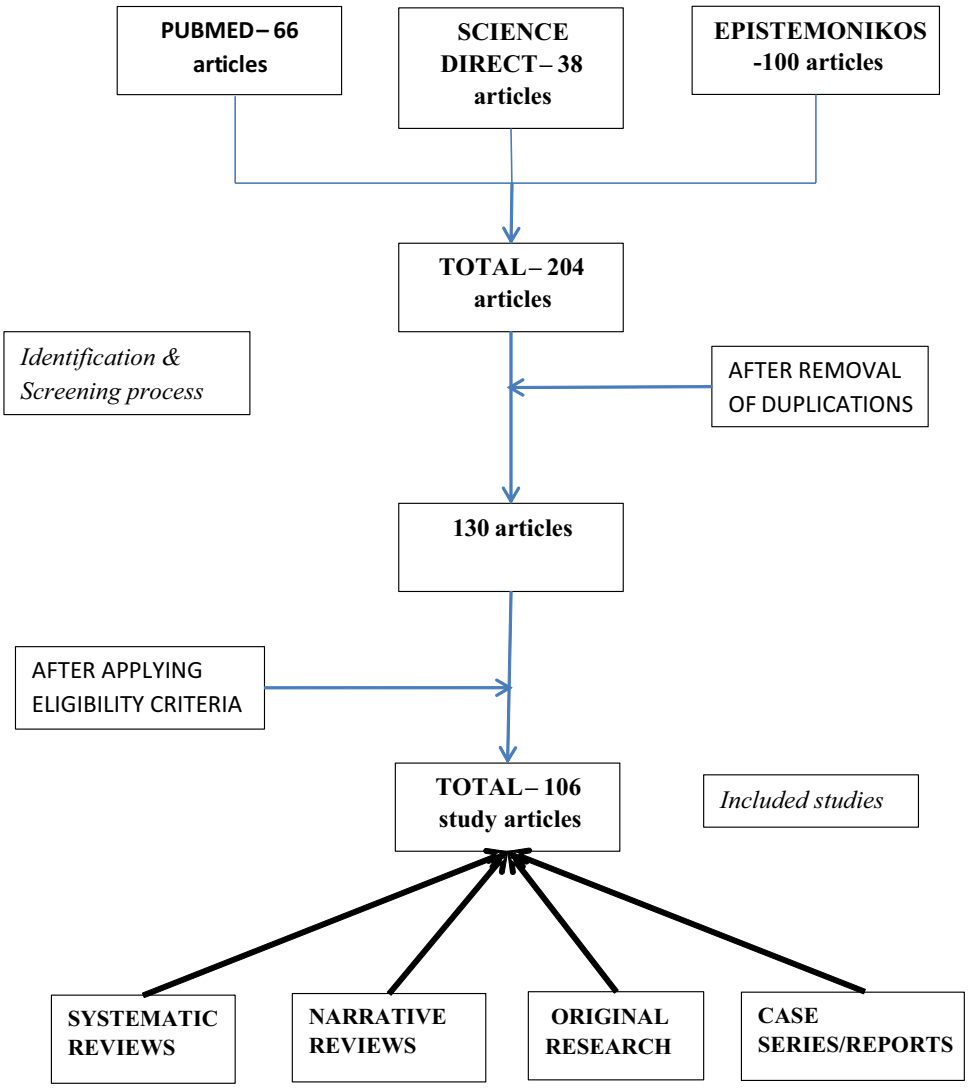

Fig. 1 Scheme showing an appraisal of the articles based on the methodology and eligibility criteria

Table 1 Findings of the study

Detection of COVID19		**Rapid test PCR positive:** Nasopharyngeal swab/oropharyngeal swab/throat swabs/bronchoalveolar lavage/sputum/trachea [3–87], Deep lung tissue swab [11, 14, 17, 21, 22, 24, 31, 35, 36, 60, 69, 70, 83, 84, 88] Heart [14, 36, 48, 69, 89]. Placenta [3, 19, 32]. Umbilical cord blood [19]. Amniotic fluid [32]. Liver, kidneys, gut [14]. Brain [24]. Rectum [24]. Testis [34]. Pleural cavity [69] **Ultrastructural examination – electron microscopy:** Lungs – trachea, alveolar cells [14, 25, 27, 27, 58, 59, 66, 68, 74, 90] Kidneys [5,34, 36, 82, 83. Heart 36, 45, 64]. Intestines [25, 27, 68]. CNS [25, 27]. Spleen, liver [25, 27]. Ovaries [27] **Molecular detection of SARS CoV2 in samples of:** Heart, lung biopsy – by targeting SARS CoV2 envelope E gene [7], SARS CoV2 nucleocapsid (N) gene [7], E gene in periodontal tissue [16], spike protein in nasopharyngeal secretion [18] **IHC:** Lung samples [12, 25, 57, 62, 68, 74, 78, 82, 84, 91–94]. Renal tubular epithelium [25, 95]. Skeletal muscle [96]. Receptor for SARS CoV2, ACE2, TMPRSS2 in airway cells [97]. Colon, CNS, liver [25] **Immunofluorescence:** Endothelial cells of myocardium sample in FFPE tissue [29] **In situ hybridization:** Alveolar cells [38, 90], virions in portal vessel lumen and endothelial cells [75] **Viral RNA detected in:** Lungs [13, 61, 76, 78, 83, 97, 98]. Cardiomyocytes [13, 78]. Olfactory nerve and brain tissue [99]. Lymph nodes, spleen [78, 98]. Eyes [100]. Pharynx, liver, bowel, kidneys, cerebral samples [78]. **Viral S protein:** [63, 101] SARS-CoV2 **IgG antibodies** by ELISA – post-mortem [15] **Endomyocardial biopsies** with histological and virological analyses positive [102], COVID19 lungs with **hyaluronan** [103] (specific detection of SARS CoV2 not available)
Comorbidities		Arterial hypertension [5, 6, 18, 21, 23, 24, 34, 35, 41, 44, 79, 88]. Diabetes mellitus [6, 18, 21, 23, 24, 35, 41, 45, 79, 88] Cerebrovascular disease, dementia, COPD, coronary artery disease, malignancy [6]. Extracorporeal life support, dialysis, pharmacological therapies given [7]. Obesity, overweight, morbid obesity [18, 21, 41, 44]. Chronic ethanol abuse [23, 41] Hyperlipidaemia, sickle cell anaemia, deep vein thrombosis [18]. End stage renal disease, illicit drug use [21]. Heart disease and lung diseases. [18, 21, 23, 31, 34, 41, 45], Kidney diseases [23, 31, 34]. Others like dementia [23], Immunosuppressive medication [24], Liver steatosis, and cerebral small vessel disease [31]. Gastric carcinoma, hepatocellular carcinoma, basal cell carcinoma [34] Graft-versus-host disease following cancer [44] Bone marrow transplantation (BMT) [44], and HIV positive status [45]
Lungs	Gross changes observed	Thrombosis and embolic phenomenon in small- and large- sized arteries [4–6, 9, 14, 17, 18, 24, 36, 40, 42, 43, 45, 46, 48, 51, 52, 59, 60, 66, 68, 70, 76, 80, 83, 86, 88, 92–94, 97, 104, 105] Pulmonary infarction, haemorrhagic infarcts [6, 27, 78, 80, 83]. Fibrosis [5, 21, 22]. ARDS [47, 62]

Table 1 (continued)

	Microscopic/histopathological changes noted	Diffuse aveolar damage – DAD [4, 6, 9, 14, 17, 20–25, 30, 31, 35, 36, 40, 41, 43, 46, 49, 50, 52, 55–58, 64, 65, 68, 69, 71, 74, 77, 78, 80, 81, 83, 86–88, 92–94, 97, 104–106] Exudative DAD seen within 8 days period. Advanced DAD seen after 17 days. Vascular involvement + in early stage of the disease [17]. Early/exudative phase DAD in < 14 days. Proliferative, fibrotic phase in median 32 days. SARS-CoV-2 + early phase [20]. Hyaline membrane formation [4, 21, 22, 24, 30, 31, 40, 52] Type 2 pneumocyte hyperplasia [4, 9, 14, 20, 22, 27, 41, 62, 66, 69, 84] Extensive inflammation [21, 95] Fibrinous plaques in alveoli [5, 21, 22] Squamous metaplasia [9, 14, 31, 45, 76, 77] Intra-alveolar and interstitial glycosaminoglycan hyaluronan (HA) localization in the exudative phase. Exudate pronounced HA staining [103]. ARDS [47, 62] Neovascularization in lungs + through intussusceptive angiogenesis with COVID19 than influenza [51]. Hemophagocytosis in pulmonary lymph nodes [71]. Galactomannan antigen positive—invasive pulmonary aspergillosis (IPA) [72]. Histiocytic hyperplasia with hemophagocytosis (HHH) [74]. Thrombi and neutrophilic extracellular traps (NETs) [33, 95]
Heart	Gross changes observed	Ventricular dilatations [6] Thromboemboli [88, 89, 92] Ischemic changes [23].
	Microscopic/Histopathological changes noted	Myocyte enlargement, nuclear polymorphism [56, 89]. Extensive inflammatory response [95, 102]. Thromboemboli [88, 89, 92]. Intracellular oedema, sarcomere ruptures, viral transcription in cardiomyocytes [13]. Myocarditis [25, 36, 56, 59, 68, 89, 91]. Ischemic changes [23]. Focal myocyte necrosis [27, 59]. Lympho-monocytic endotheliitis [29]. Interstitial mononuclear infiltrates [30]
	Enzyme markers elevated	Elevated CK (creatine kinase), CK MB, CRP (C reactive protein), D-dimer [96]
Kidneys	Gross changes observed	Thromboemboli, infarction [27, 36, 45, 88, 92]
	Microscopic/histopathological changes noted	Diffuse proximal tubular injury -necrosis 5 [6, 47, 53, 79, 92, 106]. Extensive inflammatory response [95]. Thromboemboli, infarction [27, 36, 45, 88, 92]. Marked upregulation of IL (interleukin) 6, TNF (tumour necrosis factor) alpha, IL1 beta, p38, IL8, and caspase3 in the endothelium – involved in the basement membrane disruption, endothelitis, duplication [18]
CNS	Gross changes observed	Subarachnoid haemorrhage, cortical venous thrombosis [27]. Intraparenchymal ischemia, infarcts [39, 85, 99]
	Microscopic/histopathological changes noted	Extensive inflammatory response [28, 95] Intraparenchymal intravascular microthrombi, ischemia, infarcts [22, 49, 101] Acute perivascular disseminated encephalomyelitis (ADEM) like appearance [54].
Liver	Microscopic/histopathological changes noted	Macrovesicular steatosis [6, 10, 30, 35, 40] Centrilobular necrosis [10, 87] Extensive inflammatory response [95] Hepatitis [25, 87] Platelet-rich thrombi. [83] Fibrosis of portal tract [75].

Table 1 (continued)

Placenta	Microscopic/histopathological changes noted	Mixed inflammatory infiltrates of neutrophils, monocytes in subchoroidal space and increased intervillous fibrin, funisitis [3]. Multifocal small intervillous thrombi, focal thrombosis of foetal placental vessels with foetal death at 28 weeks [19]. Patchy acute chorionitis diffuse infarction/villous necrosis [26]. Acute chorioamnionitis, placenta-massive fibrin deposition, mixed intervillitis and intense neutrophilia, lymphocyte infiltration [32].
Some pathophysiology thought to be unique in some COVID19 cases		Ferroptosis [7] IHC, monoclonal primary antibodies (anti-IL6, anti-TNF alpha, anti-ICAM (intercellular adhesion molecule) -1, anti-caspase-1 – all markers of pyroptosis) were higher [8]. Multiple aberrant immune responses involving the lungs and reticuloendothelial system [101] MCP1 (monocyte chemoattractant protein), RANTES (regulated upon activation, normal T cell expressed and secreted). IL6 and IL8 were associated with the disease progression. Severe lung damage through cell pyroptosis and apoptosis [90].
Other observations had caused significant morbidity but were not directly related to mortality		Gross observations: spleen-subcapsular infarcts [27], urinary bladder-blood vessel thrombosis [27]. Microscopic observations: haemophagocytosis, white pulp atrophy [36, 84, 98]. Infected periodontal tissue [16]. Seminiferous tubular injury, reduced Leydig cells, mild lymphocytic inflammation [34, 37].
Concurrent infections		Aspergillosis, mucormycosis [14]. Bronchopneumonia [15, 69, 83, 87]. Secondary bacterial infections [20, 23, 55]. Superimposed acute necrotizing pneumonias [24]. Respiratory viral infections [55].

Discussion

COVID19 disease in general

The COVID19 displayed heterogeneity with organ involvement and with no specific viral injury [3]. Deaths in COVID19 are multifactorial in preexisting morbidity where COVID19 acts as a contributory factor for the fatal outcome [46, 97]. All the younger patients, and other individuals having preexisting health conditions are prone to thromboembolic complications, immune dysregulation, and liver damage and other contributing influences of COVID19 than direct contribution [4, 5, 107].

A study suggested that a panel of cytokines could be used to predict disease deterioration. Severe damage was attributed to both cytopathy and immunopathologic damage [6], and worsening of the disease did not require active SARS-CoV-2 infection [7].

A case series concluded that diffuse alveolar damage (DAD), thrombosis, haemophagocytosis and immune cell depletion are the interrelated pathological processes seen in COVID19. In addition, new autopsy findings were acute pancreatitis, adrenal micro-infarction, pericarditis, disseminated mucormycosis, aortic dissection, and marantic endocarditis. Active viral replication was noted outside the respiratory tract [8]. The thrombi formation was 9 times more prevalent with COVID19 than with influenza [9] and the thrombogenicity of SARS-CoV-2 infection is linked to widespread endothelial damage [10].

In later stages of the disease course, the virus was sporadically present indicating the maladaptive immune response causing further progress in the disease and that immunomodulation should be the target of therapy [11]. Usage of steroids in the critically ill was suggested [95].

The COVID19 induced coagulopathy indicated the therapeutic measures to target the coagulopathy [12]. Other systematic reviews also documented that major findings in lungs were diffuse alveolar damage, hyaline membrane formation, and microthrombi in small blood vessels [88, 96].

There was a high incidence of deep vein thrombosis and pulmonary embolism suggestive of endothelial involvement [96]. Other organs like the heart, liver, kidney, brain, spleen, skin, and adrenals displayed inflammation and vascular damage. Massive activation of the immune system and microvascular damage were found in COVID19 [88]. DAD of the lungs was superimposed with acute bronchopneumonia. Microthrombi were described in the placenta, lungs, kidneys, and central nervous system (CNS). The gastrointestinal tract displayed minimal such changes. The endothelial injury was commonly seen in the lungs. SARS-CoV-2 viral particles were demonstrated in organ-specific cells in the trachea, lungs, liver, large intestine, kidney, and CNS [13].

Frequent testing and strict surveillance of systemic parameters were recommended owing to the rapid spread of the disease [91, 97].

LUNGS The lungs were the major target organ of severe COVID19 pneumonia with DAD and segmental pulmonary arterial thrombosis. DAD was typical exudative and proliferative phases of acute lung injury. The exudative DAD was seen within 8 days of disease and advanced stages after 17 days. The DAD occurring in COVID19 is morphologically not different from any other causes. The DAD had different phases – exudative, proliferative, and early repair phases. Hyaline membrane formation, type 2 pneumocyte hyperplasia, and acute fibrinous organizing pneumonia (AFOP) pattern were seen. There were multinucleated giant cells, smudge cells and vascular thrombosis present [14–25, 99, 101, 102].

SARS-CoV-2 viral particles primarily damaged the type 2 pneumocytes and caused thrombophilic activity [26–28]. Organotropism was highly seen in the lungs and the reticuloendothelial system [29].

The virus initiates direct damage in the acute phase and gets cleared by the body's immune response in the organizing phase. The period of presence from infection is approximately 10 days, absent in the organizing phase of the disease [30, 31]. Viral RNA was found in the lungs and virus particles in endothelial cells and pneumocytes. The early onset pro-inflammatory, activation of the complement pathway/coagulation cascade resulting in systemic procoagulant state and endothelial expression of cytokines and alveolar macrophage infection by SARS-CoV-2 lead to cytokine storm and thrombotic microangiopathy, as well as damage to multiple organs [22, 32, 33].

The COVID19 is a unique disease characterized by extensive lung thrombosis and long-term viral RNA persistence in pneumocytes and endothelial cells – with the presence of infected cell syncytia [34]. In a few there was massive bilateral alveolar damage in the early acute respiratory distress syndrome (ARDS) which correlated with severe disease onset and progressive deterioration leading to death. Type 2 pneumocyte hyperplasia with atypia was seen. There were no inclusion bodies present [35]. SARS-CoV-2 was detected in airways and pneumocytes by immunohistochemical staining and electron microscopy for virions [36]. In a study the virus was detected in alveolar macrophages but not in extrapulmonary tissues [103]. Interestingly an observation was made on the presence of coronavirus-like particles in the kidneys and GIT other than the respiratory system by immunohistochemistry and ultrastructural examination using electron microscopy [37]. Co-infections like respiratory viral and bacterial infections were seen [103]. Coagulopathy in the lungs like pulmonary artery embolism and overall deep vein thrombosis was present [20, 36, 38].

The deaths that occurred in the second week were related to SARS-CoV-2 pneumonia. The deaths occurring earlier were noted from heart failure and those occurring later were due to complications [39].

Radiologically the ground glass appearances seen in postmortem CT dependent portions were nonspecific; whereas ill-defined round opacities, traction bronchiectasis and reverse halo sign can be considered key findings in COVID19 [40]. In some, lungs showed features of global multifocal reticular consolidation in the postmortem CT [41, 42].

The response to systemic thrombolysis was low because of inflammatory and prothrombotic changes in the arterial wall resulting in a lack of lung perfusion [43]. The deceased who were treated for a longer duration did not show capillaritis, vasculitis or endotheliitis, but thrombosis was majorly present showing advanced stages of organization. Unique finding obtained in a study was the presence of invasive mycosis and florid pneumonia in the areas of patchy DAD, and the presence of aspergillosis, and mucormycosis [44]. On the contrary, there was no evidence of invasive aspergillosis in a set of critically ill patients [45].

In one study, severe secondary bacterial infections were seen in the diabetic patients and also severe illness in preexisting disease conditions [89]. The treatment of COVID19 patients should also aim at the secondary acute bronchopneumonia and aspiration pneumonia developed during the course [21, 46].

Heart In the heart the findings observed were inconsistent. Some displayed direct injury of the heart by the virus, some did not. The COVID19 cases frequently had cardiac fibrin microthrombi but there was no evidence of direct myocardial infection [47]. In one study less than 50% of SARS-CoV-2 were detected in the myocardium causing cardiac dilatation, ischemia, and mural microthrombi [48]. Individual case studies reported that the SARS-CoV-2 causes fulminant myocarditis[49, 50], on the contrary, one case report ruled out the virus causing the same [51].

The concept of direct injury of the heart and lungs and the procoagulant stage created by the virus was supported by a study [52].

A study solely focusing on the heart revealed that COVID19 leads to small vessel endotheliitis in the heart [53]. In another study [103], there was no extrapulmonary tissue-specific virus finding but the cardiac diseases were aggravated during COVID19 in a cohort [33], whereas in a study in children, cardiac dysfunction with COVID19 was due to myocardial stunning/oedema associated with the systemic inflammatory state and direct myocardial injury by SARS-CoV-2, and hypoxia was attributed as secondary to viral pneumonia. [54].

Brain Data obtained indicated that SARS-CoV-2-related brain injury may be due to several pathogenetic mechanisms or due to direct viral effects [55]. A case demonstrated the CNS complications in COVID19 patients, providing potential parainfectious processes affecting the patients [98]. The neuropathological changes were mild and found no evidence of brain damage directly by SARS-CoV-2 [56]. There were various hypoxic-related neuropathological changes in the brain but no neurotropism was seen [90]. However, a study demonstrated that cerebrovascular accidents can be associated with COVID19 [57].

Kidneys The data provided evidence of direct kidney injury – the presence of a cluster of coronavirus particles in the tubular epithelium with upregulation of ACE2 receptors causing acute kidney injury (AKI) [58, 100]. However, the AKI was mild, suggestive of potential reversible kidney damage [59].

Muscle The skeletal muscle displayed damage by the viral cytopathic effect and by elevated cytokines [60].

Teeth SARS-CoV-2 was demonstrated in the periodontal tissue [61].

Testes The testes exhibited significant seminiferous tubular injury, reduced Leydig cells and mild lymphocytic inflammation; however, there was no evidence of SARS-CoV-2 virus in the testes [62] Supporting it there was impaired spermatogenesis in COVID19 and in addition occurrence of autoimmune orchitis [63], suggesting precautions in the process of sperm donation.

Liver COVID19 is not associated with any liver-specific histopathology [64] Aiding to it, a study confirmed that liver failure was not the main target of COVID19. Whatever derangement of the intrahepatic blood vessel network displayed, it was secondary to systemic changes caused by the virus [65].

Eyes As analysis of three different sequences – RdRo-gene, E-gene, and Orf1 gene – the existence of SARS-CoV-2 viral RNA was proved to be present in the retina. [66].

Fetus A spontaneous miscarriage occurring in a primigravida who was positive for SARS-CoV-2 in the nasal, placental, umbilical cord, and amniotic fluid swabs during labour did not rule out local bacterial infection [67]. Similarly, there was a case of foetal demise in a woman confirmed with SARS-CoV-2 infection without any other causes in COVID19 pregnancies [68]. In one study confirmative foetal death as an outcome of SARS-CoV-2 infection in pregnancy was displayed with direct infection of SARS-CoV-2 on the placenta [92].

In HIV patients In the HIV-positive patient, a study[69] did not show any specific findings with COVID19.

Some of the unique findings obtained which are applicable in general

- Ferroptosis was the proposed cause of ischemia–reperfusion injury in COVID19 causing cardiac and multiple organ injury [70].
- The endothelial dysfunction and pyroptosis pathway were the cause of systemic thrombotic events [71].
- The organ damage was due to extensive NETs and vascular damage [11, 72].
- HA (glycosaminoglycan hyaluronan) was demonstrated in the alveolar spaces in the COVID19 lung in lethal cases. Advised adjuvant therapy targeting HA may be used in treating COVID19 [73].
- Viral RNA was detected even in formalin-fixed paraffin-embedded tissues of the lungs, airways, lymph nodes, and spleen [93].
- Platelet-rich fibrin microthrombi play a significant role in systemic coagulopathy [74].
- SARS CoV-2 is associated with haemophagocytic lymphohistiocytosis (HLH). Identification of HLH will be useful for therapeutic strategies [75].
- Demonstrated galactomannan antigen – confirmed invasive pulmonary aspergillosis (IPA). Recommended further assessment of the frequency of occurrence of IPA [76].
- Lesions of histiocytic hyperplasia with haemophagocytosis (HHH) in the majority of cases demonstrated that COVID19 triggers a systemic immune-inflammatory disease [77].

Conclusions

As displayed by various studies, the effect of SARS-CoV-2 is heterogeneous. It affected highly the lungs, followed by the heart in its direct effect. Other organs including the brain, kidney, skin, skeletal muscle, eyes, and testes were seen affected by the disease. The liver and gastrointestinal tract were minimally affected. The disease progression was rapid and more deteriorating in the patients having preexisting medical conditions. In this review, there were ferroptosis, pyroptosis, NETs and platelet-rich fibrin microthrombi identified as unique pathogenetic mechanisms. The presence of histiocytic hyperplasia with haemophagocytosis (HHH), hemophagocytic lymphohistiocytosis (HLH), and glycosaminoglycan hyaluronan (HA) in lethal cases was observed. The occurrence of secondary bacterial infections and invasive pulmonary aspergillosis should be considered in the therapeutic approach. Special attention should be given to the COVID19 pregnancies. It is observed that a full autopsy

cannot be replaced by minimal autopsy techniques. The studies also supported that a forensic pathologist should always be suspicious of the pathogen in the pandemics while conducting any medicolegal autopsy.

The concerning factor is that the findings are so heterogenous in the organs involved and the pathology seen that the definitive course of the disease cannot be decided in an individual who has developed COVID19.

Key points

1. The lungs followed by the heart are greatly affected directly by the virus.
2. Disease progresses rapidly in patients having preexisting medical conditions.
3. This review obtained information about ferroptosis, pyroptosis, neutrophil extracellular traps (NETs) and platelet-rich fibrin microthrombi as unique pathogenetic mechanisms in COVID19.
4. The presence of histiocytic hyperplasia with hemophagocytosis (HHH), haemophagocytosis lymphohistiocytosis (HLH) and glycosaminoglycan hyaluronan (HA) in lethal cases was observed.
5. Secondary bacterial infections and invasive pulmonary aspergillosis should be considered in the therapeutic approach.

Author contribution Dr. Raviraj KG contributed to the conception and design, acquisition of data and analysis, and interpretation of data. Dr. Shobhana SS contributed to the planning and tabulation needed for the review.

Declarations

Ethics approval Not applicable.

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