Review Article

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The Biochemistry and Pathology Characteristics of Coronavirus Disease 2019

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Abstract

Coronavirus Disease 2019 started appeared in December 2019 China's, Hubei Province, Wuhan. During the following three months, it spread rapidly to numerous provinces in China and transversely the world. The initial bunch was epidemiologically associated with a seafood wholesale market in Wuhan. Many essential aspects of the pathogenesis, pathology, and pathophysiology of SARS- CoV2 have not yet clear. Here, we offer a comprehensive overview of the biochemistry Characteristics and histopathological results present in various organs and cells and the pathophysiology. In this review we found many damage in some organic system, such as pulmonary edema with hyaline membrane formation, desquamation and hyperplasia of pneumocytes, proteinaceous in Respiratory tract, spleen atrophic, degeneration and focal necrosis of hepatocytes, Esophageal, stomach and bowel in digestive tract. Degenerated or necrosed myocardial cells in Heart. Degeneration and shedding of renal tubules epithelial cells, hyaline casts in Kidneys and other organs.

Keywords: SARS- CoV2, Histopathology, Pathophysiology, Biochemistry, Lung.

1. Introduction

Severe acute respiratory syndrome 2 (SARS- CoV2) started emerged in China's Hubei Province in December 2019 in Wuhan. During the following three months, it spread rapidly to many provinces in China and across the world. The initial cluster was epidemiologically associated with a seafood wholesale market in Wuhan. However, many of the first 41 cases had later reported having no known exposure to the market [1]. The WHO raised the COVID-19 to pandemic worldwide, March, 2020, it seems to be very contagious and has quickly spread worldwide. This virus COVID-19 belongs to a family of large, enveloped, positive, and single-stranded RNA viruses that cause SARS and MERS [2-4]. It isolated from lower respiratory tract samples of 4 cases [5], COVID-19 infection typically causes severe manifestations related to the lower respiratory tract [6]. COVID-19 has a characteristic clinical, where patients had shown like to other coronavirus symptoms, counting fever, fatigue, muscle ache, cough, and dyspnea, whereas diarrhea, confusion, headache and vomiting were rare [7-10]. The pathological characteristics of SARS - CoV2 closely resemble those detected in MERS and SARS coronavirus infections [11]. The effective pathological outcome in these cases of patients with SARS - CoV2 was diffuse alveolar damage (DAD) [10]. This severe lung injury and others organs such as liver, in cases with COVID-19 is caused by both direct viral effects and immunopathogenic factors [12]. Many essential aspects of the pathogenesis, pathology, and pathophysiology of COVID-19 have not yet clear. Here, we offer a comprehensive overview of the biochemistry and histopathological results present in various organs and cells and the pathophysiology.

1.1 Clinical Biochemistry Characteristics of Severe acute Respiratory Syndrome 2

Life pattern of COVED-19 in host cells starts with the authoritative of S proteins to cell receptor Angiotensin converting enzyme 2(ACE2). Subsequent to bound, the adaptation of S protein changed promotes viral envelope combination with cell layer through endocytosis pathway. ACE2 is one of the major receptors for SARS-CoV and SARS-CoV-2 [13,14].The white blood cell

contain was lower than ordinary in 9% of patients, and neutrophil include expanded in 38% of patients. Lymphocytes and hemoglobin in numerous patients were underneath the typical range. Numerous patients similarly created unusual myocardial protein profiles with expanded creatine kinase (CK) and expanded lactate dehydrogenase (LDH). A few patients demonstrated anomalous liver capacity in changing degrees, indicating expanded alanine aminotransferase (ALT) or aspartate aminotransferase (AST). A few patients likewise created differing degrees of renal hindrance, indicating expanded blood urea nitrogen (BUN) or serum creatinine (Cr) [7]. Of the 9 patients, 5 had lymphopenia (< 1.0×109/L) and 3 had raised ALT and AST, of which 1 had an ALT level of 2093 and a degree of 1263 U/L [13].

Numerous biomarkers have been related with helpless results and speak to a contender for hazard definition models for foreseeing serious SARS-CoV-2 so as to direct clinical consideration. Among all, lymphopenia, thrombocytopenia, leukocytosis, CRP, PCT, LDH, AST, ALT, D-dimer, cTn represent the most prescient boundaries of extreme SARS-CoV-2 [15].

1.2 The pathology and pathophysiology of severe acute respiratory syndrome 2 Pathology

There is currently a lack of pathology that has been described since the first epidemic in December 2019 on COVID-19, biopsy, or autopsy. Although, the coronavirus disinfection in histopathological laboratories. It appears reasonable to refrain from working frozen sections on potential cases of COVID-19 except the laboratory is certain in curbing aerosols in the cryostat; and make confirmed that formalin fixation and paraffin embedding should inactivate COVID-19 [16]. The pathological findings of Certain human organs of SARS-CoV-2, such as the Lungs, Liver, Heart and other organs, are imperfectly described. For ease of reference, the primary pathological findings for each organ summarized in Table 1. These pathological characteristics of SARS-CoV 2 show essential similarities to SARS-CoV and MERS-CoV infection [10, 15, 17, 18].

1.3 Pathophysiology

Pathophysiology and virulence mechanisms of Coronavirus, and hence also of COVID-19 have connections to the function of the non-structural proteins (nsps) and structural proteins. For example, research underlined that nsps could block the host innate immune response [19] , thus founds immune dysfunction is a common characteristic in cases of COVID-19 that might be an essential factor correlated with disease severity and mortality [14]. Early studies on SARS-CoV and MERS-CoV infection there have shown that increased amounts of proinflammatory cytokines in serum SARS patients (e.g., IL1B, IL6, IL12, IFNγ, IP10, and MCP1) [20], and MERS patients (e.g., IFNγ, TNFα, IL15, and IL17) [21], were associated with pulmonary inflammation and general lung damage [20-23]. SARS-CoV2 infection also reported had high amounts of cytokines in patient's serum (e.g., IL1B, IFNγ, IP10, GCSF, MCP1, MIP1A, and TNFα) [1]. Moreover, patients requiring ICU admission had higher amounts of some cytokines than did those not requiring ICU admission, suggesting that the cytokine storm was associated with disease severity [1]. However, COVID-19 infection, probably leading to activated T-helper-1 (Th1) cell responses, also initiated increased secretion of T-helper-2 (Th2) cytokines (e.g., IL4 and IL10) that repress inflammation, which varies from SARS-CoV infection [1, 20].

As know, Coronaviruses are the envelope, positive-stranded RNA viruses. Between the functions of structural proteins, the envelope has a crucial role in virus pathogenicity as it promotes viral attachment and release. Amongst the fundamental components of Coronaviruses, there are the spike glycoproteins composed of two subunits (S1 and S2). Of note, in COVID-19, the S2 subunit, including a fusion peptide, a transmembrane domain, and cytoplasmic domain, is highly conserved. The dysfunction results as a result of the dysregulated host response to infection by COVID-19, signs dysfunction include severe dyspnea, low oxygen saturation, impaired renal function, reduced urine output, tachycardia, acute heart injury, hypotension, cold extremities, skin mottling, abnormal liver function and altered mentation [1, 8, 24, 25]. Besides, 5% of patients can develop severe disease with features of respiratory failure, cardiac injury, septic shock, Raetia, or multiple organ dysfunction [24, 26].

Organs	Pathology changes	References
Respiratory tract	Diffuse alveolar damage showed pulmonary edema with hyaline membrane formation, desquamation and hyperplasia of pneumocytes, proteinaceous, Interstitial mononuclear inflammatory infiltrates, dominated by lymphocytes, multinucleated syncytial cells with atypical enlarged pneumocytes characterized by large nuclei, amphophilic granular cytoplasm, and prominent nucleoli were identified in the intra-alveolar spaces, showing viral cytopathic-like changes.	[6, 25, 27]
Immune System	Spleen: Spleen atrophic, Macrophage's proliferation and phagocytosis, Focal hemorrhage and necrosis. lymph nodes: decreased number of lymphocytes and focal necrosis. Decreases of CD4+ and CD8+ T-cell levels, and abnormal cytokine levels in the spleen and lymph nodes. Bone marrow: Myelopoiesis is decreased in bone marrow.	[14]

Digestive tract	Liver: Micro vesicular steatosis and mild lobular and portal activity, a few interstitial mononuclear inflammatory infiltrates, degeneration and focal necrosis of hepatocytes. Esophageal, stomach and bowel: Degeneration, necrosis and desquamation of epithelium mucosae of variable degree. The gallbladder is prominently distended.	[6, 14, 27, 28]
Heart and blood vessels	Heart: A few interstitial mononuclear inflammatory infiltrates, Degenerated or necrosed myocardial cells. Blood vessels: Shedding of endothelial cells, end vasculitis and thrombi.	[10, 27]
Central nervous system	Degeneration of some neurons, neurological manifestations, Cerebral hyperemia and edema, SARS-CoV-2 causes neuroinflammation & induces Lewy body formation in the brains of macaques.	[29, 30]
Kidneys	Kidneys: Degeneration and shedding of renal tubules epithelial cells, and hyaline casts. Microthrombi and fibrotic foci are found in the kidney interstitium.	[27]
Glands	Necrotic foci are noted in the adrenal glands.	[27]

Table 1: The pathological changes in different organs and tissue

COVID-19 is a systemic disease that can move beyond the lungs by blood-based dissemination to affect multiple organs. These organs include the kidney, liver, muscles, nervous system, and spleen. The early cell-based portal for viral entry is through the angiotensin-converting enzyme 2 receptor.

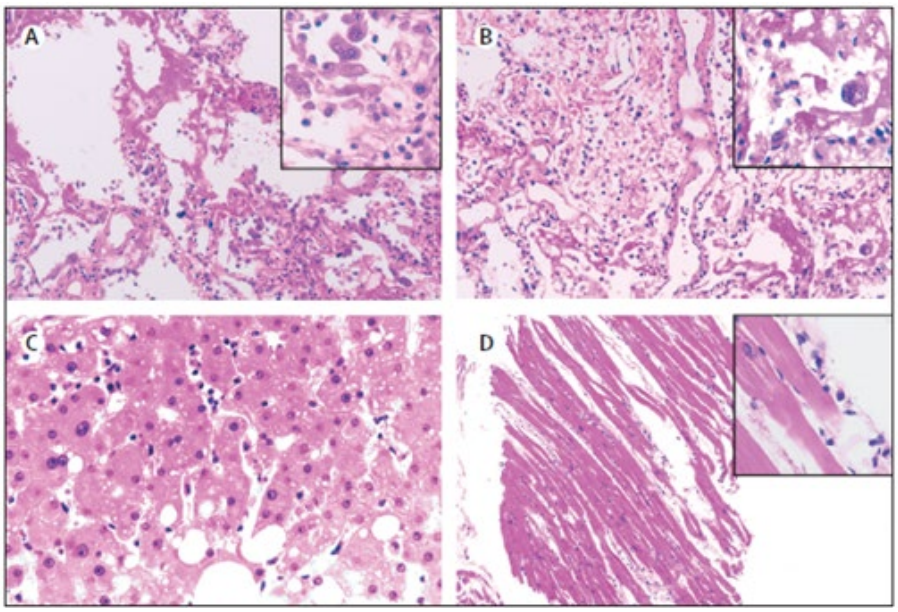

Figure 1: Pathological Manifestations of Right (A) And Left (B) Lung Tissue, Liver Tissue (C), And Heart Tissue (D) In A Patient With Severe Pneumonia Caused By Sars-Cov-2[6].

SARS-CoV-2=severe acute respiratory syndrome corona virus 2.

2. Conclusion

In conclusion, COVID-19 epidemics can occur anywhere and the indication designated that this novel Coronavirus has an etiologic role in severe acute respiratory syndrome. This new virus appears to be very transmissible and has quickly spread worldwide. SARS-CoV-2 has been shown to infect human respiratory epithelial cells and lung tissue, also damage Liver, stomach, duodenum, and rectal mucosa in patients. Furthermore, the Immunopathology may also play a pertinent role in the development of disease harshness.

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