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Research Article

Biochemical and Demographical study in patients with corona virus

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Abstract

Coronavirus disease (COVID-19) is an infectious disease caused by the SARS-CoV-2 virus. Most people infected with the virus will experience mild to moderate respiratory illness and recover without requiring special treatment. However, some will become seriously ill and require medical attention. Older people and those with underlying medical conditions like cardiovascular disease, diabetes, chronic respiratory disease, or cancer are more likely to develop serious illness. Anyone can get sick with COVID-19 and become seriously ill or die at any age. The objective of this study is to evaluate of some biochemical test and their complication in patients with COVID-19. The practical part included cross-sectional study and collecting any information and tests about the 49 patients with COVID-19 and 55 as a control in Karbala province. The obtained data were classified according to sex, age, mortality besides the biochemical tests. The results of this study showed that there is an increment in the concentration of urea and CRP comparing with the normal range as well as the level of WBC. In addition, the results revealed that the percentage of covid-19 patients in male was more than in female and the percentage of infected of age over 45 years was high. Furthermore, the D-dimer of 88% of patients was over 250 ng/ml. Besides, the mortality rate of male (57%) was more than in the female.

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1- Introduction

Many new illnesses have developed in recent years across a variety of geographical regions, with pathogens like the Ebola virus, the Zika virus, the Nipah virus, and coronaviruses [1]. A novel CoV strain (2019-nCoV) has just been identified in Wuhan City, China, and is currently known as severe acute respiratory syndrome[2]. Initial genomic sequencing data of this virus do not correspond with previously sequenced CoVs. Covid 19[3]. While coronavirus disease 2019 (COVID-19) is thought to have originated in an animal host (zoonotic origin) before spreading to humans, additional paths should not be ruled out[4]. COVID-19 has a less severe etiology but stronger transmission competence than previously identified human CoVs, as evidenced by the rising number of confirmed cases worldwide[5]. COVID-19 has a low pathogenicity and intermediate transmissibility when compared to other emerging viruses such as Ebola virus, avian H7N9, COVID-19, and Middle East respiratory syndrome coronavirus (MERS-CoV) [6]. According to research on codon use, this unique virus likely originated in an animal source like a bat Real-time PCR and next-generation sequencing have early pathogen detection through early diagnosis [7].

A cluster of individuals with unexplained viral pneumonia was discovered in Wuhan, China, in December 2019[8]. Several tests were performed to determine the disease's etiological agent, and as a result, the severe acute respiratory syndrome coronavirus (COVID-19), the Middle East respiratory syndrome coronavirus (MERSCoV) [8], and other common respiratory viruses were ruled out. Ultimately, scientists determined that a new coronavirus known as severe acute respiratory syndrome coronavirus 2 was to blame (COVID-19). On March 2020, the World Health Organization (WHO) proclaimed the coronavirus disease 2019 (COVID-19) to be a pandemic due to a sharp rise in the number of afflicted persons [10]. Around 145 million people have been infected with COVID-19, and over 3 million have died as a result of the disease. As COVID-19 is a novel infection for humans, it is essential to create safe and efficient vaccination techniques to effectively stop the pandemic and restore normalcy[15].

1-1 Evolution of the Coronavirus

Because of the crown-like spikes on their surface that can be seen when studied under an electron microscope, coronaviruses get their name from the Latin word corona, which means "crown" or "halo. The biggest known RNA viral genome is that of the coronavirus, an enclosed virus with a non-segmented, single-stranded, positive-sense RNA genome of about 32 kilobases. Coronaviruses are members of the nidovirales order's coronavirinae subfamily of the coronaviridae family[12]. The COVID-19 strain is categorized within the betacoronavirus genus according to the genome sequence analysis[9]. The Coronavirinae subfamily has four genera: alphacoronavirus, betacoronavirus, deltacoronavirus, and the gammacoronavirus. As the coronavirus genome is known to have a 5′ cap and a 3′ poly (A) tail, it functions as an mRNA for translation of the replicase polyproteins necessary for viral replication once the coronavirus has infected the host cell[21]. According to reports, animal reservoirs including bats, mice, rats, chickens, dogs, cats, horses, and camels are where most coronaviruses are thought to live, similar to the 2015 Zika virus outbreak, the virus has recently gained the potential to spread among humans through zoonotic transmission and start an epidemic[11].The fact that so many bats concentrate in one area and have the capacity to fly great distances makes the crossing of the animal-human species barrier more likely. Bats have been reported to be the principal vector and reservoir for a wide range of viruses, including the coronavirus. The first human coronaviruses were identified in the 1960s[13]. There are now seven distinct human coronavirus strains known through investigations. Mild respiratory tract infections are known to be brought on by the four prevalent coronavirus strains, including 229E, NL63, OC43, and HKU1. Formerly recognized

coronaviruses that infected animals may mutate and adapt to infect people, leading to the formation of a new virus and the potential for a pandemic epidemic [14]. Examples of viruses that are known to induce more severe symptoms in patients include the SARS-CoV, MERS-CoV, and the more recent COVID-19. These viruses are known to cross the animal-to-human species barrier. the mortality rate reveals that MERS-CoV and SARS-CoV were more likely to result in death in an infected individual, despite the fact that the number of COVID-19 positive cases has far outpaced the number of MERS-CoV and SARS-CoV cases [16].

In general, because of their replication strategy and the absence of viral RNA poly

1-2 Structure of COVID-19

Large, non-structural polyproteins (ORF1a/b) are encoded by the COVID-19 genome. These polyproteins are further broken by proteases to produce 15–16 proteins, 4 structural proteins, and 5 accessory proteins (ORF3a, ORF6, ORF7, ORF8, and ORF9) (Figure below). [18] The four structural proteins, which are necessary for COVID-19 assembly and infection, are the spike (S) surface glycoprotein, the membrane (M) protein, the envelope (E), and the nucleocapsid (N) protein. Spike's ability to bind to host cells is mostly dependent on the spike surface glycoprotein, which can be further broken down by host proteases into an N-terminal S1 component and a membrane-bound C-terminal S2 section. When the S1 subunit binds to a host

merase proofreading activity, RNA viruses, including the coronavirus, influenza virus, and HIV, are known to have exceptionally high mutation rates. The fundamental unit of evolution, mutations enable natural selection for characteristics that are advantageous to the virus, such as increased virulence, adaptability, and evolvability. Phylogenetic analysis performed by [16]. According to reports, COVID-19 spread from bats purchased at the Huanan South China Seafood Market in Wuhan, Hubei Province, China, over the species barrier. Also, the study found that COVID-19's genomic sequence shared a higher degree of similarity with the SARS-like bat coronavirus RaTG13 (96.2% identity) than it did with either the SARS-CoV or the MERS-CoV (51.8%), suggesting that bats are the major source of COVID-19 [17].

receptor, the prefusion trimer can become unstable, causing the S1 subunit to shed and the S2 subunit to change into a very stable postfusion conformation. [19] The S1 subunit's receptor-binding domain (RBD) engages a host receptor by undergoing hinge-like conformational motions that momentarily conceal or reveal the receptor binding characteristics. The "down" conformation and the "up" conformation can be thought of as the two states of the S1 subunit. The latter corresponds to an accessible state of the receptor whereas the former indicates an inaccessible state of the receptor. In order to create monoclonal antibody medications and to direct the design and development of vaccinations, it is therefore beneficial to understand the structure and function of the spike protein. [22]

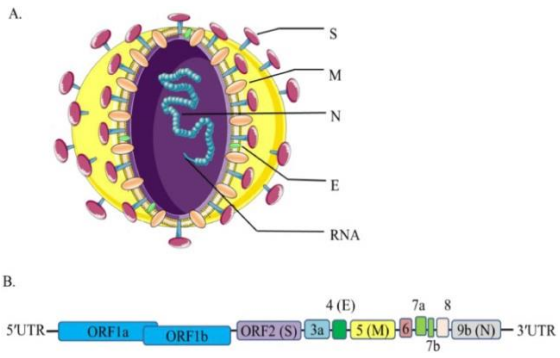

Figure (1-1) Structure and genome of severe acute respiratory syndrome coronavirus 2 (COVID-19).

In above figure: (A) There are four structural proteins as follows: spike (S) surface glycoprotein (purple); membrane (M) protein (orange); nucleocapsid (N) protein (blue); and envelope (E) protein (green). Genomic RNA is shown encased in the N protein. (B) The COVID-19 genome is arranged in the order of 5′-replicase (ORF1a/b)–structural proteins [spike (S)–envelope (E)–membrane (M)–nucleocapsid (N)]−3′.

1-2-1 S Glycoprotein

A large, multipurpose class I viral transmembrane protein is the coronavirus S protein. IBV, or infectious bronchitis virus, in chicken causes the size of this abundant S protein to range from 1,160 amino acids to 1,400 amino acids (FCoV, feline coronavirus). It rests in a trimer on the virion surface, giving it the appearance of a corona or crown. Functionally, it is necessary for the infectious virion particles to engage with different host cellular receptors and enter the cell [20].

Moreover, it plays a crucial role in the determination of host range and tissue tropism. Importantly, S protein is one of the essential CoV immunodominant proteins that can activate host defense mechanisms. The two S1 and S2 subunits that make up the ectodomains of all CoVs S proteins are organized similarly. S1 assists in binding to the host receptor, while S2 is responsible for fusion. The N-terminal domain (NTD) and the C-terminal domain are further separated into the first (S1) (CTD). These two subdomains function as receptor-binding elements that effectively communicate with a variety of host receptors. The receptor-binding motif is present in the S1 CTD (RBM). The trimeric S1 positions itself on top of the trimeric S2 stalk in each coronavirus spike protein. [23]

1-2-2 M Protein

The M protein gives the viral envelope a distinct shape and is the most prevalent viral protein in the virion particle [30]. It interacts with the nucleocapsid and serves as the main coordinator of coronavirus elongation [24]. Although the amino acid composition of coronavirus M proteins is very variable, they nonetheless have a lot of structural similarities across various genera. The M protein comprises three transmembrane domains, each of which is flanked by a long carboxy terminus and a short amino terminus [25]. Overall, the viral scaffold is maintained by M-M interaction. Of note, the M protein of COVID-19 does not have an amino acid substitution compared to that of SARS-CoV [26]

1-2-3 E Protein

Among all the important structural proteins, the coronavirus E protein is the tiniest and most mysterious. It functions in a variety of ways throughout the pathogenesis, assembling, and release of the virus. It functions as a viroporin and is a tiny integral membrane polypeptide (ion channel). [27] Due to morphological and tropism alterations, the inactivation or lack of this protein is associated with altered coronavirus pathogenicity. The E protein is made up of three domains: a C-terminal domain that is effective, a long hydrophobic transmembrane domain, and a short hydrophilic amino terminal domain. Same amino acid composition is seen in the COVID-19 E protein without any substitutions. [28]

1-2-4 N Protein

Coronavirus N protein has a variety of functions. Among its many roles, it helps M proteins interact during virion assembly, which is necessary for complex formation with the viral genome, and improves the virus's transcription efficiency. It has three different and highly conserved domains: an NTD, an RNA-binding domain (also known as a linker region, or LKR), and a CTD. The NTD is significantly divergent in both length and sequence, and it attaches to the 3′ end of the viral genome, possibly by electrostatic interactions. The charged LKR is often referred to as the SR (serine and arginine) domain because it contains a lot of serine and arginine. The LKR is in charge of cell signaling and has a direct interaction capability with in vitro RNA interaction [29].

1-3 Causes

Coronavirus illness 2019 is brought on by infection with the novel coronavirus (severe acute respiratory syndrome coronavirus 2, or

COVID-19) (COVID-19), the virus that humans, and new information on how it does so is always being uncovered. According to data, people in

close proximity are the ones who are most likely to contract it (within about 6 feet, or 2 meters). The virus spreads by respiratory droplets produced when an infected person breathes, sings, talks, coughs, or sneezes. A

1-4 Stages of the disease

There are three phases of COVID-19 infection, with each stage become more severe:
Stage I: The initial phase of viral infection or viral response, when upper respiratory tract infection symptoms predominate.
Stage II: The respiratory phase, during which patients experience full-blown pneumonia and all of its symptoms.
Stage III: The period of hyperinflammation during which patients have ARDS, sepsis, and failures of the kidneys and other organs.

1-5 Diagnosis

The two most typical symptoms of COVID-19 are fever and cough, while some patients may also have sputum production, sore throat, headache, myalgia/arthralgia, rhinorrhea, and diarrhea. As a condition progresses to pneumonia, it causes shortness of breath and discomfort. As a result, abrupt anosmia or ageusia may serve as a clinical screening tool to identify COVID-19 patients. Of note, a sizable percentage of patients reported olfactory and gustatory problems. The majority of patients exhibited lymphopenia, raised C-reactive protein, normal or reduced leukocyte counts, and some also had thrombocytopenia,

1-6 Biochemical Monitoring of COVID-19 Patients

Beyond etiological COVID-19 identification, clinical labs play a crucial role in this epidemic. It is crucial to monitor the biochemical status of COVID-19 patients using in vitro diagnostic tests in order to evaluate the severity and course of the illness as well as the efficacy of treatment intervention. Numerous widely used in vitro diagnostic tests have been linked to adverse COVID-19 development and may offer crucial prognostic

causes COVID-19 spreads quickly among person close may inhale these droplets or they may end up in their mouth, nose, or eyes in some situations, the COVID-19 virus can spread by a person being exposed to small droplets or aerosols that stay in the air for several minutes or hours — called airborne transmission.[29]

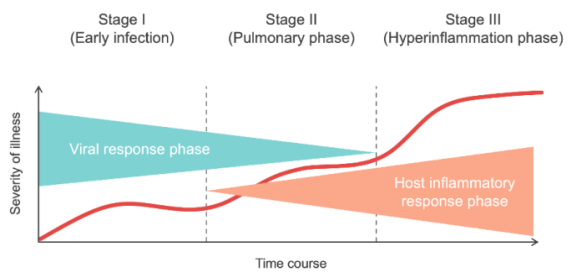

Figure (1-2) phases of COVID-19 infection

high D-dimer, lactate dehydrogenase, and alanine aminotransferase. On chest computed tomography (CT) scans of patients with pneumonia, ground-glass opacity is a common radiological finding that can be difficult to identify on chest X-rays. In patients with severe pneumonia, local or bilateral patchy consolidation has also been found on CT images. To confirm the diagnosis, viral diagnostic techniques specific to COVID-19 should be used because these clinical, laboratory, and imaging findings are vague and cannot distinguish COVID-19 from other viral respiratory infections[32].

data. The key laboratory abnormalities connected to adult COVID-19 patients and their probable clinical indication are listed below, along with a recommended test list based on recent literature. This research reveals that individuals with severe COVID-19 may also be at risk for cytokine storm syndrome in addition to more typical laboratory findings. If feasible, cytokine testing, in particular IL-6, should be utilized to evaluate severe patients who may be suffering from hyperinflammation [7] [23].

Testing for COVID-19 entails examining samples to determine whether the virus is present or has ever been. The virus or antibodies created in response to infection are detected by the two major branches. To identify specific cases and enable public health officials to track down and manage outbreaks, molecular tests for the presence of viruses through their molecular constituents are utilized. Instead, antibody testing (serology immunoassays) reveal a person's past exposure to the illness. Since antibodies may not emerge for weeks after infection, they are less effective for identifying ongoing illnesses. The determination of the infection fatality rate is aided by the use of this method to determine disease prevalence [21]

1-7 Molecular test (aka RNA or PCR test)

A real-time reverse transcription polymerase chain reaction (rRT-PCR) test is the COVID-19 RT-PCR Test. The test may be performed using a singleplex format (three separate assays for each of the three COVID-19 targets) or multiplexed into a single reaction with an amplification setup, which includes all three COVID-19 targets. The test employs one primer and probe set to identify human RNase P (RP) in a clinical sample and three primer and probe sets to detect three areas in the COVID-19 nucleocapsid (N) gene in a singleplex format. The test employs two primer and probe sets to identify two areas in the COVID-19 N gene and one primer and probe set to detect RP when multiplexed into a single response. Sputum, lower respiratory tract aspirates, bronchoalveolar lavage, nasopharyngeal wash/aspirate, and nasal aspirate are examples of upper and lower respiratory specimens from which RNA is recovered. These specimens are then reverse transcribed to cDNA and amplified. The forward and reverse primers are where the probe anneals to a particular target sequence during the amplification process. The attached probe is degraded by Taq polymerase's 5' nuclease activity during the PCR cycle's extension phase, which causes the reporter dye to dissociate from the quencher dye and produce a fluorescent signal. QS7 measures fluorescence intensity throughout each PCR cycle.[32] [46]

1-7-1 Rapid Antigen Test

The COVID-19 Antigen Rapid Test (Nasopharyngeal Swab) is a quick, cassette-based chromatographic immunoassay for the qualitative detection of COVID-19 antigens in nasopharyngeal swab samples from people who have a suspected COVID-19 infection in conjunction with clinical presentation and the outcomes of other laboratory tests. In upper respiratory specimens collected during the acute phase of coronavirus infection, COVID-19 antigens are often detected.[33]

Positive findings indicate the presence of coronaviral antigens, however to assess the level of infection, clinical correlation with patient history and other diagnostic data is required. Good outcomes do not rule out bacterial illness or viral co-infection. The substance found might not be the actual disease-causing agent. Negative findings do not rule out COVID-19 infection and should not be the only factor considered when making decisions about the patient's care or course of therapy. [34]

Negative findings should be considered presumptive and, if necessary for patient treatment, verified with a molecular analysis. Negative findings should be taken into account in light of a patient's history, recent exposures, and the existence of clinical symptoms and indications that are compatible with COVID-19.[35]

1-8 Epidemiology

Three essential components are required for an infectious illness to emerge: an infectious source, a transmission pathway, and a vulnerable population. COVID- As 19 infected people create a lot of virus in the upper respiratory tract before symptoms appear, they are the main source of viral transmission. The majority of infected people are asymptomatic and continue with their daily routines, which causes the virus to spread quickly and covertly [31]. Asymptomatic infections have the potential to spread since the viral load seen in asymptomatic people is identical to that of symptomatic patients. At the start of the epi-

demic, COVID-19's basic reproductive number (R0), which is used to define an infectious agent's transmissibility, was 2.97. Aerosol droplets, close contact, and maybe fecal or oral transfer are the main methods of COVID-19 person-to-person transmission. Even though COVID-19 viral RNA was discovered to be stable on the surface of plastic and stainless steel in an experimental setting, studies looking into the infectious potential of inanimate objects and patient remains revealed that they were not contaminated by a live virus, indicating that contact transmission is unlikely to happen via contaminated surfaces. Patients with COVID-19 infections had lower fatality rates and severity levels than those with SARS and MERS, but COVID-19 is far more prevalent and contagious than SARS and MERS.[5] [21]

1-8-1 Coronavirus Versus Seasonal Influenza

Every year, influenza, or the seasonal flu, affects everyone in the world; it typically strikes between December and February. Because it is not a reportable infection (and therefore does not need to be reported to the municipality), it is hard to estimate the number of reports each year because patients frequently do not seek medical attention for mild symptoms. The Rate of Case Fatality was recently estimated at 0.1%.[21]

An estimated 3-5 million cases of severe influenza are reported each year, and between 250,000 and 500,000 people die worldwide. In the majority of affluent countries, those over 65 account for the bulk of fatalities. Also, it is dangerous for women who are expecting, young children under the age of 59, and those who have life-threatening conditions. [36]

In most underdeveloped nations, the yearly immunization prevents illness and serious hazards, although it is nevertheless a known yet painful element of the season.Contrary to seasonal influenza, coronavirus is less prevalent, has resulted in fewer cases to far, has a higher case fatality rate, and lacks an effective treatment. Despite the high rate, there have been fewer linked deaths as a result of the constrained blowout from person to person. [37]

1-8-2 Coronavirus Versus Common Cold

The most prevalent ailment that affects humans is the common cold; on average, an adult will have two to three colds every year, while a child will get six to eight colds in the same amount of time. Although there are more than 200 different kinds of cold-associated viruses, infections are infrequent, deaths are exceedingly rare, and they often affect people who are very elderly, very young, or who have compromised immune systems.[32]

1-9 Effect of COVID-19 on Organs

Evidence shows that COVID-19 may influence other sections of the body in addition to respiratory disorders, which may be related to pneumonia, sepsis, or lung failure. The figure below shows some typical, unusual, and severe symptoms seen by COVID-19 patients.[16]

1-9-1 Effect of COVID-19 on kidneys

Even patients with no prior history of renal disease but who are now battling severe COVID-19 infections are displaying evidence of kidney damage. According to early studies, up to 30% of COVID-19 patients hospitalized in China and New York suffered from moderate to severe renal damage. According to reports from New York physicians, the proportion may be higher. [38]

High amounts of urine protein and abnormal blood tests are indicators of renal issues in COVID-19 patients. In certain situations, the kidney damage is serious enough to call for dialysis. Several hospitals reporting spikes in COVID-19 patients with severe illness have reported running low on the equipment and sterile fluids required to carry out these kidney treatments. [39]

Angiotensin converting enzyme 2 (ACE-2) receptors are the virus' first stop when it enters cells to begin the process of infection. These receptors are found in the cell membranes of the tissues that line the heart, arter-

ies, lungs, kidneys, and gastrointestinal system. By controlling angiotensin levels, a protein that elevates blood pressure by tightening According to certain studies, COVID-19 may target the kidneys more frequently than other organs since the cells lining the proximal tubule have a very high level of ACE-2 expression. The majority of the reabsorption of water and nutrients from the blood occurs in the proximal tubule, a significant kidney section. [41]

1-9-2 Effect of COVID-19 on liver

According to reports of a global meta-analysis to evaluate the incidence and severity of liver illness in people with severe and non-severe COVID-19 infections, liver damage is more frequently linked to severe than nonsevere disease. The severe COVID-19 group has higher mean values of ALT, AST, and bilirubin than the nonsevere group. This is consistent with the high ALT/AST values that 16-53% of patients had at the start of the epidemic. Moreover, individuals with severe COVID-19 have been reported to have a seven-fold increase in indirect indicators of liver damage, such as hypoalbuminemia, and a 1.7-fold increase in the incidence of hyperbilirubinemia.[43]

The wide spectrum of symptoms linked to COVID-19 might be explained by the virus' affinity for ACE2, which is expressed on several human cell types. Since ACE2 is expressed in both liver cells and bile duct cells and pathological studies in patients with SARS infection discovered in 2003 reveal the presence of the virus in liver tissue, it is possible that liver damage in patients with COVID-19 infection may be directly caused by the viral infection of liver cells. The impact of antibiotics and antiviral medications given to patients as well as the cytokine storm associated with infection may potentially contribute to the explanation of the liver enzyme abnormalities [44]. Moreover, the rise of hepatic ALT/AST may be influenced by underlying pre-existing liver conditions. Thus, the existing COVID-19 therapies, particularly the use of steroids, may encourage the reactivation of latent chronic hepatitis B infection, a major contributor to liver disease. So, in order to manage infection effectively, doctors need take into account all of these criteria. [45]

1-9-3 Effect of COVID-19 on heart

Several studies suggest that, like some viral infections, 2019-nCoV infection may be associated with cardiac damage. According to a research conducted on 400 hospitalized patients in Wuhan, China, around one-fifth of COVID-19 patients acquired cardiac illness, which raised the patient death rate. Arrhythmias are brought on by severe and abrupt inflammation of the heart muscle, which also reduces the heart's capacity to pump blood effectively [46]. Patients who have a history of cardiovascular disease and high blood pressure are therefore more likely to die than healthy people The lining of the heart and blood arteries is harmed by oxygen shortage brought on by lung injuries. In addition, fever and inflammation may cause fatty plaques in blood vessels, they assist in managing blood pressure. [40]

Once within kidney cells, COVID-19 starts to reproduce utilizing the tools of the cell. During this process, cells frequently experience damage. After the immune system detects the viral invaders, it also triggers an inflammatory reaction. This reaction could unintentionally injure healthy tissue further.[42]

the arteries of the heart of persons with or without signs of cardiovascular disease to become unstable, which can result in vascular blockage and cardiovascular issues. Atypical blood coagulation and venous thromboembolism are other potential illnesses that may be present in COVID-19 hospitalized patients, necessitating the treatment of anticoagulants or thromboprophylaxis for these individuals. These issues may become more severe in these circumstances due to the release of numerous inflammatory cytokines. Hence, cytokine inhibitors could be useful for lessening the severity of the illness.[51]

1-9-4 Effect of COVID-19 on lungs

COVID-19 has the potential to result in lung problems such pneumonia and, in the

most severe instances, ARDS. Another potential COVID-19 consequence, sepsis, can injure the lungs and other organs permanently. [47]

With pneumonia, the lungs swell and swell with fluid, making breathing harder. Some people's breathing issues might get so bad that they need hospital care with oxygen or possibly a ventilator.The pneumonia brought on by COVID-19 often affects both lungs. Shortness of breath, coughing, and other symptoms are brought on when the fluid-filled air sacs in the lungs restrict the ability of the organs to absorb oxygen. Although most people recover from pneumonia without any long-term lung damage, COVID-19-related pneumonia can be quite serious. Lung damage may cause breathing problems that persist even after the sickness has passed and may take months to resolve. [48]

1-10 Prevention of Covid-19

The WHO has indicated that the crucial measures in controlling infectious illnesses like COVID-19 include education, isolation, prevention, regulating the transmission, and treating sick individuals. Making the following suggestions can help to reduce the spread of illness. [49]

Shielding is the practice of avoiding direct contact with any healthy (perhaps asymptomatic patients) or diseased person while remaining at home (home quarantine); avoiding unnecessary travel; adhering to the two-meter rule for social distance in crowded areas, especially if someone is sneezing or coughing; refraining from shaking hands while introducing yourself to people; Washing hands frequently for at least 20 seconds with soap and water or using hand sanitizer containing at least 60% alcohol is advised, especially after touching communal areas, using the restroom, or shaking hands. Avoid touching your eyes, nose, or mouth with unwashed hands, and disinfect surfaces with household sprays or wipes. [50]

It should be noted that employing a medical mask (particularly N95) or a respirator (specifically FFP3) should be advised owing to the lengthy incubation period and the existence of asymptomatic individuals. Also, it has been advised to sterilize the used respirator, reuse it just once, and properly dispose of the used masks. Many techniques, such as steam, hydrogen peroxide, or radiation, might be taken into consideration for the sterilization of worn masks in light of the significant contamination of respirators and surgical masks during the COVID-19 pandemic. In addition to being advised to utilize medical shields or protective garments, especially for healthcare workers, routine hand washing is the greatest defense against COVID-19 infection.[41] [47]

2 -The Practical Aspect:

The work process started from Imam Al-Hussein Specialized Hospital (the epidemiological stone lobby) in period from 28/12/2020 to 10/2/2021 for collecting information about the infected patients, as they had difficulty accessing the census rooms due to the privacy laws applied by the hospital. In order to preserve the privacy of patients and because of the seriousness of the existing health situation at the time, we were able to access the information of the quarantined patients exclusively and directly, as each patient was asked about his name, age, and all the information that would help in completing the research. They were sorted according to gender, age, and the most important analyzes that the patient underwent, in addition to the fact that the number of infected patients inside the halls is small in relation to the percentage of swabs that were taken from patients, and this indicates that many patients quarantined themselves inside the house. At the conclusion of the research, we got the following results:

Table 1: The level of biochemical parameter in patients with covid-19 comparing with normal range

Parameters	Mean ±SD(n=75)	Normal range
Hb (g/L)	12.8 ± 2.7	12.5-17.5
WBC (X10 ^{9})	12.07 ± 6.11	4.5-11
Urea (mg/dL)	57.96 ± 39.83	7-20
Creatinine (mg/dL)	0.92 ± 0.91	0.74 ± 1.35
CRP (mg/L)	12.45 ± 28.4	Less than 10
ALT (U/L)	31 ± 16.74	7-55
AST (U/L)	40.94 ± 18.17	5- 40

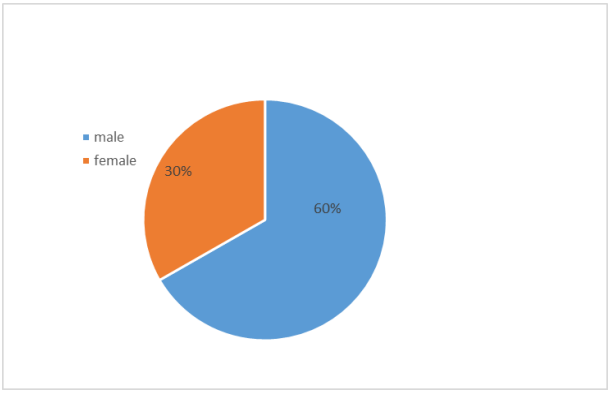

Figure (2-1): The percentage of patients with covid-19 according to the sex

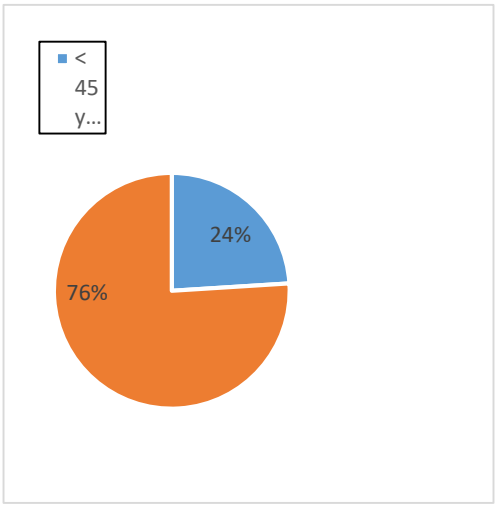

Figure (2-2): The percentage of patients with covid-19 according to the age

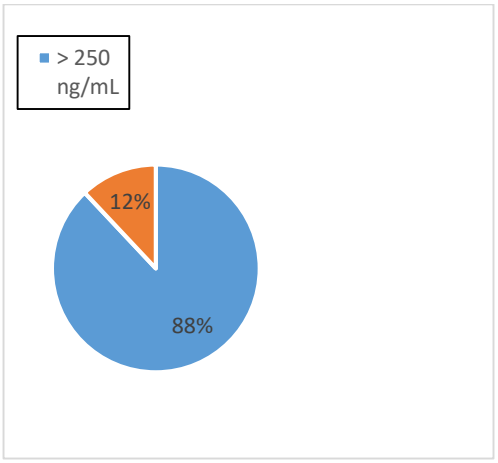

Figure (2-3) : The level of D-dimer in patient with covid-19

Table 2: The percentage of covid-19 patients with normal and abnormal level of biochemical tests

Parameters	Percentage % of patients	
	Normal	Abnormal
Hb (g/L)	60	40↓
WBC (X10 ^{9})	59	41↓
Urea (mg/dL)	9	91↑
Creatinine (mg/dL)	98	20↓
CRP (mg/L)	86	14↓
ALT (U/L)	93	70↓
AST (U/L)	54	46↓

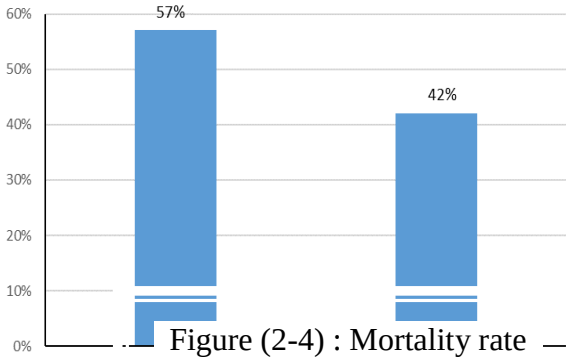

Figure (2-4) : Mortality rate of male and female

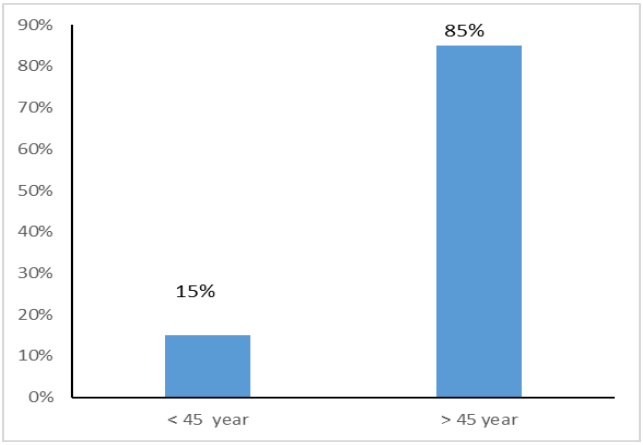

Figure (2-5) : Mortality rate according to the age

3. Discussion

High blood urea, as shown in Table No. (1), is due to several reasons. Doctors noticed a high number of kidney failure cases among people infected with the Corona virus. In critical cases of Corona virus disease, the blood clots were created faster, which may lead to the formation of small clots that block the blood vessels. This can also happen in the kidneys, which is what some experts call "small clots in the kidney tissue." In addition, there is scientific evidence that the virus may directly attack the kidneys.

In the event that Corona patients develop pneumonia and need artificial respiration, the matter affects the kidneys in a significant way. The pneumonia caused by Corona leads to a large accumulation of water around the lungs. In this case, doctors give the patient diuretics that help withdraw fluids from the body, which requires the kidneys to work more than normal, while they suffer from a lack of blood flow due to small clots, which accelerates their failure.[35]

The higher incidence of infection for males compared to females, as shown in figure (2-1) is due to the fact that

The emerging corona virus uses the angiotensin ACE2 enzyme to penetrate the cells of its victims. The enzyme is secreted more in men than in women, and ACE2 is found in many parts of the body, including the heart, lungs and kidneys. The Corona virus exploits this

enzyme to penetrate the body. The immune system of women is stronger than that of men. The female hormone “estrogen” stimulates the immune system, while the male hormone “testosterone” suppresses the immune system. This is why the immune system of women reacts faster than men. Men are more susceptible to risk factors that cause disease. Men smoke more than women, and men consume more alcoholic beverages compared to women.” Smoking and consumption of alcoholic beverages are among the most prominent risk factors for disease. The high coagulation rate of the injured shown in figure (2-3) is due to several reasons, the most important of which are [47]:

The clotting occurs when the “emerging corona virus” attacks the endothelial cells lining the blood vessels, as the virus does this by binding to the ACE2 receptors located in the endothelial cell membrane, and once it binds to the receptors, the blood vessels release proteins that cause blood clotting That "Covid-19" causes the body's immune system to provoke an overactive inflammatory response, and this inflammation may cause blood clotting, The presence of a certain level of a protein in the blood of the patient called “von Willebrand VW.” This protein is synthesized in endothelial cells and platelets, and its main function is to form a framework for platelet adhesion. It is known that it is stored in vascular endothelial cells, and once some damage is caused by the corona virus, blood clotting, in which the protein plays an important factor in its occurrence, occurs, and it is a complex process during which the blood forms clots, and it is worth noting that the level and activity of this protein in the blood of humans differs markedly between healthy people.

Patients with severe COVID-19 infection showed two times higher rate of abnormal liver function tests compared to those with moderate COVID-19 infection, indicating possible liver damage, as well as severe respiratory symptoms (main effect for COVID-19 infection). Furthermore, patients with hepatic function problems showed higher levels of inflammatory cytokine and serum chemokines, a feature of the cytokine storm associated with severe COVID-19, compared to those with normal liver function of clinical significance for hepatic dysfunction associated with COVID-19. The extent of its prevalence is not entirely clear due to the scarcity of available studies on the subject. From here, scientists are trying to understand whether these complications are caused by the disease itself, or as a result of other factors such as the body's inflammatory reaction or drug complications. Systemic viral infections are often associated with a temporary elevation of liver biomarkers resulting from immune activation without impaired liver function, a phenomenon known as transient hepatitis. Currently prescribed medications used to relieve some of the severe symptoms of COVID-19 can stress the liver in people with the virus who have severe symptoms, especially for patients who already have pre-existing liver problems [1]. Moreover, hepatopathy may be significantly exacerbated by taking medications such as antivirals (Hydroxychloroquine, Lopinavir/Ritonavir, and TCM), antibiotics, antipyretics and pain relievers. Patients with cirrhosis of the liver and those with COVID-19 may be exposed to an increased risk of developing acute and chronic liver failure, and this explains the high levels of ALT and AST.

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