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The Association of Super Infection with SARS-COV2 Virus Infection

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Abstract: *The literature on the association of Co-infection and Super infection in patients with SARS-COV2 infection is reviewed in this paper. Co-infection is defined by the Centers for Disease Control and Prevention in the United States as an infection that occurs in the human body when the person is already afflicted with multiple diseases. Super infection is another phrase used by the Centers for Sickness Control and Prevention to describe a disease caused by the same virus but with a different strain each time. Over a lengthy period of time, it has been seen that a patient's viral respiratory infection develops to bacterial infection, which can be fatal. However, the association between the two remains a mystery for the researchers. In the case of COVID-19 viruses, comparable results have been obtained. The extensive spectrum of Co-infections and Super infections in Corona patients has been documented in a number of articles.*

Keywords: SARS-COV2

I. INTRODUCTION

It has been observed over time that respiratory illnesses lead to bacterial infections, which impact the patient's immunity either directly or indirectly. Many investigations in the disciplines of microbiology and epidemiology have been conducted in this regard. The influenza virus infection was the subject of one such study. According to the study, secondary bacterial infection caused the bulk of mortality during the 1918-9 influenza pandemic.This infection had a large influence on pregnant women where a fetal pneumonia was a common symptom.The data from the influenza pandemics in 1957 and 1968 isn't as extensive. The information gathered is useful not only during a pandemic or epidemic, but also for diagnosing and treating diseases, as well as research into medications and vaccines for the pathogen. The recommended treatment for influenza-related community-acquired pneumonia (CAP) is to treat bacterial CAP with empiric antibiotics at the same time. After 48–72 hours, this treatment can be stopped, especially if no bacterial co-pathogens are found.Several investigations were conducted during a previous H1N1 pandemic, and it was discovered that secondary bacterial infection occurred often in patients. Symptoms of various respiratory viruses and bacteria were also included. Death rates were extremely high.

II. REVIEW OF LITERATURE

2.1.Corona Virus Infection

Humans are the most common careers for coronavirus or COVID-19. The infection is directly related to upper lung infections in adults and several other lung infections in both children and adults. COVID-19 pneumonia arose in Wuhan, China (Fro). The initial scientific name was for COVID virus was 2019n-COVand later it was changed to SARS-COV-2. It is related to different types of pneumonia:i) Middle East Respiratory Syndrome (MERS)and Severe Acute Respiratory Syndrome(SARS).No case of Co-infection or Secondary infection was reported.

Considering the studies of previous viruses, a question related to co-pathogen arose. Answering this question was considered the most important task because COVID-19 patients were already given certain medications in the hospital. Another important question that arose was whether the COVID-19 is a Co-infection or Super infection.Following that, the COVID-19 management released early guidelines for doctors to treat suspected corona patients with antibiotics as soon as feasible. According to several research papers and articles, only 8% of all patients were considered Co-pathogens for Covid-19. Asthma, bronchitis, diabetes, and high blood pressure were all present in these patients.

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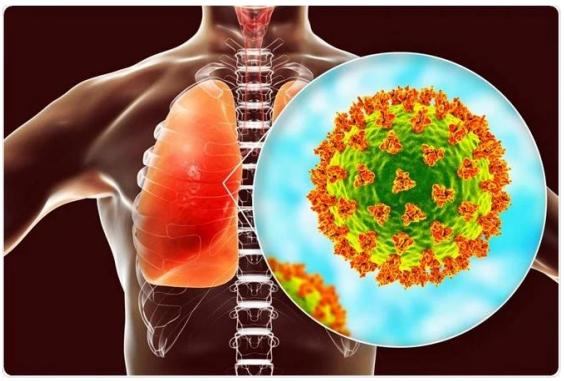
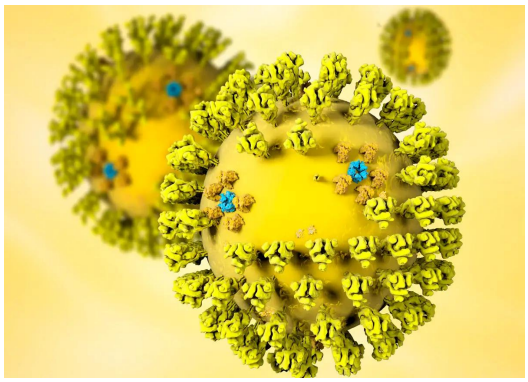
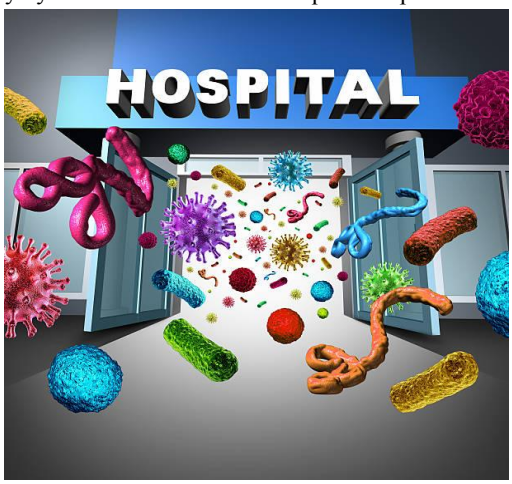




2.2 Possible Methods for Spreading Infection

Infections can be acquired mainly by two methods: Hospital Acquired and Community Acquired.

"If the illness is detected after 48 hours of hospitalisation, it is acquired from the hospital; if the infection is identified before 48 hours of hospitalisation, it is community acquired," according to the Centers for Disease Control and Prevention (CDC) in the United States. Once a patient was diagnosed with an infection, he or she was admitted to the hospital. [1] This was thought to be an instance of secondary/super infection as well as co-infection. The Centers for Disease Control and Prevention (CDC) later categorised superinfections as illnesses that develop after a previous infection, particularly when caused by germs that are resistant to or have become resistant to the antibiotics used earlier. Bengochea and Banford have published numerous articles describing secondary bacterial/super infection and co-infection caused by COVID-19. [7] However, the publications are unclear as to whether the COVID-19 is a Co-infection or a Superinfection. Another topic addressed in the essay is the existence of infection in the patient. The experts are still undecided as to whether the patient's infection was a real infection or viral carriage. The article's main goal was to provide a quick overview of the relationship between Co-infection and Super infection, which is caused primarily by bacterial infection and requires a specific antibiotic therapy to cure corona patients.



2.3 Antimicrobial stewardship

Both excellent and terrible antimicrobial resistance were observed during the COVID-19 pandemic. The positive aspects of this pandemic were the use of masks, preserving social distance, less crowds in public areas and public transportation, the recommendation of hand sanitizers and hand washes, isolation of the diagnosed patient, and maintaining a sterile atmosphere. Overuse of antibiotics and "repurposed drugs" without the presence of coinfection in the diagnosed patient was a detrimental side effect noted during the pandemic. Only 3% of people treated to hospitals in the United States had co-infection caused by coronavirus, according to a study conducted in the United States. Various

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attempts have been made to check for the presence of Co-infection and Super infection in the COVID-19 cases, with some hospitals and research centres in the United States being targeted. [7] Similar attempts were conducted in Wuhan, China, where four of the patients had secondary infection signs in the ICU. [2]

2.4 Clinical Data for Co-infections of Coronavirus

The study mentioned below a based on COVID-19 and its identification. The study also proves the presence of super and Co infection in the patients.

2.4.1 Case Report

Many cases of COVID-19 co-infection followed by other respiratory disorders caused by viral and bacterial infections were documented in the review literature. [8] Ozaras and colleagues described a situation in which six individuals were infected with coronavirus and had influenza Co-infection at the same time. Later, these six individuals were sent for a specialised Co-infection screening, and it was discovered that just a couple of them had COVID-19 Co-infection. Co-infection symptoms ranged from minor to severe, depending on the instance. [6] As a result, the symptoms were not recognised sooner. Other cases have also been documented in which individuals were diagnosed with pneumococcus co-infection. Cucchiari and colleagues reported five cases of corona infection, two of which were suspected and three of which were confirmed by PCR testing for pneumococcus super infection. Following the review of these articles, it was discovered that the pathogen for Co-infection is detected in corona patients who have symptoms of concurrent pneumococcal infection, as confirmed by a urine test.

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GOVT. OF NCT OF DELHI
Delhi State Health Bulletin _COVID -19
(No. 401/ April 9th 2021)

- **COVID-19 Positive Cases Status of last 24 hours**

Positive Cases	8521
Tests Conducted	109398
Positivity Rate	7.79 %
Recovered/Discharged/Migrated	5032
Deaths	39

- **COVID-19 Patient Management**

	Total beds	Occupied	Vacant
Hospital	9342	4732	4610
Dedicated COVID Care Centre	5525	122	5398
Dedicated COVID Health Centre	82	68	14
Home Isolation	13188		

* 05 beds of CCC are occupied by persons under quarantine including travelers who came By Vande Bharat Mission and Bubble flights

- **COVID-19 Testing Status**

RTPCR/CBNAAT/TrueNat tests conducted today	70403
Rapid antigen test conducted today	38995
Total tests done so far	15366581
Tests per million	808767

- **COVID-19 Positive Cases Status : Cumulative**

Cumulative Positive Cases	706526
Cumulative Positivity Rate	4.6 %
Recovered/Discharged/Migrated	668699
Deaths	11196
Case Fatality Rate	1.58 %
Active Cases	26631

- Total Number of Containment Zones as on date: 4768
- Calls received in Control Room: 1140
- Total number of calls dispatched to ambulances: 1769
- Total number of calls refused: Nil

- Hon'ble Lt. Governor of Delhi chaired a meeting of Delhi Disaster Management Authority to discuss COVID-19 pandemic situation in NCT of Delhi.
- Union Home Secretary chaired a meeting to review the requirement of DRDO Hospital in NCT of Delhi in view of recent surge in COVID-19 cases.
- An order has been issued for escalation of COVID beds in two Delhi Government Hospitals (GTB hospital and Lok Nayak Hospital)
- An order has been issued to 115 private hospitals to reserve 50% of their total ICU & Ward bed capacity or 1.25 times the occupancy on 08.04.2021, whichever is higher.

All figures are tentative, subject to change on receipt of additional information

2.4.2 Case Series

Several case studies were performed in countries like France, Iran, The United States of America, UK and Spain. After reviewing these articles, it is still not clear COVID-19 it is a true Co-infection or super infection. The following are the primary reasons for this ambiguous indication: -

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1. These studies indicate that these symptoms of the infection were seen during the hospitalization of the patient.
2. We are not clear with the timing of the collection of microbial specimens of the patient.
3. We do not know whether the Co pathogen who is isolated or not.

The majority of infections found in these research were compatible with being hospital-acquired. [9] Because of differences in the type of specimen collected, the type of test done, and the pathogen panel used, these studies are not directly comparable.

TABLE: Demographic and clinical characteristics of the patients enrolled.

	All	In Home Isolation (a)	Hospitalized (b)	p-Value a vs. b
N° of patients	40	24	16	
N° (%) of patients in class:				
15–29	4 (10)	3 (13)	1 (6)	0.63
30–39	2 (5)	2 (8)	0	0.50
40–49	11 (28)	7 (29)	4 (25)	1.0
50–59	8 (20)	6 (25)	2 (13)	0.43
60–69	8 (20)	6 (25)	2 (13)	0.43
>70	7 (17)	0	7 (44)	0.0006
Age, years, median (IQR)	52 (41.25–65.75)	43.5 (39.75–55.25)	69 (48.5–80.25)	0.0017
N° (%) of males	20 (50%)	14 (58)	6 (38)	0.33
N° (%) of patients with comorbidity:	22 (55)	10 (42)	12 (75)	0.054
Arterial Hypertension	17 (42)	5 (21)	12 (75)	**0.0011**
Diabetes Mellitus	4 (10)	1 (4)	3 (19)	0.28
Malignancy	4 (10)	0	4 (25)	**0.019**
Chronic Respiratory Disease	4 (10)	1 (4)	3 (29)	0.28
Cardiovascular Disease	8 (20)	4 (17)	4 (25)	0.69
Renal Insufficiency	2 (5)	1 (4)	1 (6)	1.0
Symptoms, N° (%) of subjects:	37 (92.5)	21 (88)	16 (100)	1.0
Fever	31 (77)	17 (71)	14 (88)	0.27
Cough	15 (37)	6 (25)	9 (56)	0.093
Dyspnea	5 (13)	0	5 (33)	**0.0066**
Anosmia	12 (30)	6 (25)	6 (38)	0.48
Ageusia/Dysgeusia	13 (33)	8 (33)	5 (32)	1.0
Diarrhea	8 (20)	2 (8)	6 (38)	**0.042**
Nausea	3 (8)	1 (4)	2 (13)	0.55
Lack of appetite	23 (58)	15 (63)	8 (50)	0.52
Fatigue	24 (60)	15 (63)	9 (56)	0.75
Myalgia	23 (58)	15 (63)	8 (50)	0.52
Rhinorrea	2 (5)	1 (4)	1 (6)	1.0
Conjunctivitis	2 (5)	0	2 (13)	0.15
Skin lesions	2 (5)	1 (4)	1 (6)	1.0
N° (%) of patients with CT evidence of interstitial pneumonia	14 (35)	0	14 (88)	**<0.00001**
Time, days elapsed from the first positive swab to negative swab, median (IQR)	22.5 (13.75–32)	22.0 (12.2–32.0)	22.5 (17.5–32.7)	0.75

2.4.3 COVID-19 co-infection in children

Only a few research have been undertaken to determine whether COVID-19 is co-infected in children. COVID-19 infection in children differs from COVID-19 infection in adults, including clinical, epidemiological, and radiological differences. COVID-19 functions as a Co-infection in paediatric patients, causing pneumonia in the lower respiratory tract.

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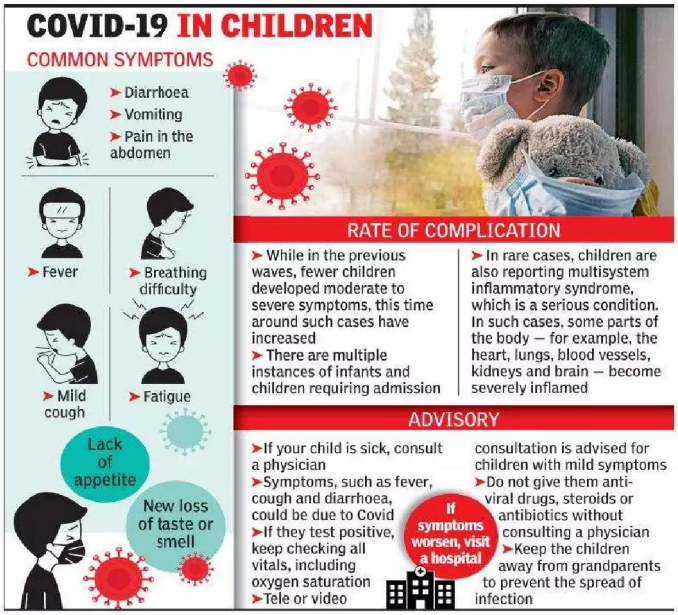

TABLE: Clinical characteristics of asymptomatic infections and other types of COVID-19

Type	Clinical characteristics	RT-PCR test for COVID-19
Asymptomatic	No clinical symptoms and chest imaging findings.	Positive
Mild	Mild clinical symptoms, such as fever, fatigue, cough, anorexia, malaise, muscle pain, sore throat, dyspnea, nasal congestion, headache. No abnormal chest imaging findings.	Positive
Moderate	Mild or moderate clinical features. Chest imaging showed mild pneumonia manifestation.	Positive
Severe	Suspected respiratory infection symptoms, plus any of the following: Shortness of breath, RR ≥ 30 breaths/min; At rest, oxygen saturation ≤93%; $Pa0_2/Fi0_2 \leq$ 300 mmHg (1 mmHg = 0.133 kPa). Chest imaging showed the lesions significantly progressed > 50% within 24–48 h was a severe disease.	Positive
Critical	Rapid progress of disease, plus any of the following: Respiratory failure, and need mechanical ventilation; Shock; Combined with other organ failure requires ICU monitoring treatment.	Positive

RT-PCR, reverse transcriptase-polymerase chain reaction; RR, respiratory rate; $Pa0_2$, arterial partial pressure of oxygen; $Fi0_2$, oxygen concentration; ICU, intensive care unit.

TABLE: Laboratory report of children suffering with Covid-19

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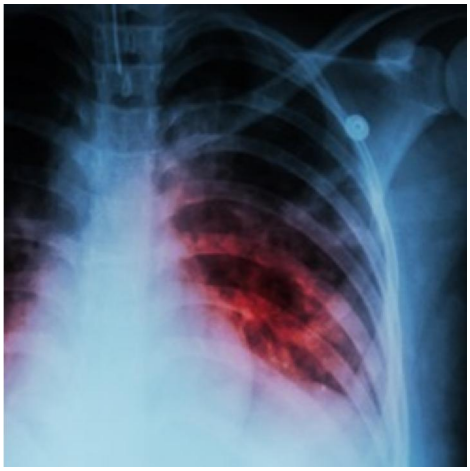
Characteristic	Normal range	Category of patients						P value	
				With clinical symptoms or/and CT abnormalities					
		All patients (n=32)	Asymptomatic infection (n=6)	Total (n=26)	Only clinical symptoms (n=7)	Only CT abnormalities (n=5)	Both (n=14)	a	b
Age and sex									
Age (year)	–	8.9±4.7	7.8±3.7	9±5.1	8.6±5.1	9.8±4.1	8.9±5.7	0.477	0.913
Sex (M/F)	–	17/15	2/4	15/11	4/3	5/0	6/8	0.341	0.094
Routine blood tests									
Leucocytes ($\times10^9$/L)	3.5–9.5	6.1±2.3	8.6±4.0	5.5±1.1	5.6±1.4	5.0±0.7	5.6±1.1	0.011	0.620
Neutrophils ($\times10^9$/L)	1.8–6.3	3.0±2.0	4.9±3.8	2.5±1.1	3.0±1.6	1.9±0.7	2.5±0.7	0.006	0.227
Lymphocytes ($\times10^9$/L)	1.1–3.2	2.6±1.1	3.1±1.5	2.5±1.0	2.2±0.9	2.7±0.7	2.5±1.2	0.233	0.591
Platelets ($\times10^9$/L)	125–350	257±103	345±116	237±91	247±89	152±115	263±69	0.082	0.056
Haemoglobin (g/L)	–	132±11	131±4	132±12	130±12	142±8	130±13	0.753	0.236
Blood biochemistry									
ALT (U/L)	0–45	25±19	28±16	25±20	22±12	29±21	25±23	0.693	0.848
AST (U/L)	0–45	33±17	34±8	33±19	30±10	34±6	34±25	0.896	0.871
Albumin (g/L)	40–55	45.3±3.1	47.5±3.2	44.8±2.9	45.6±2.8	45.7±2.2	44.0±3.1	0.071	0.379
LDH (U/L)	120–250	289±157	313±116	284±166	380±247	213±7	257±124	0.709	0.180
TB (μmol/L)	1.7–21	11.5±9.6	7.3±2.7	12.4±10.3	20±13.6	7.8±2.4	10.3±8.6	0.243	0.067
D-dimer (μg/mL)	0–0.5	0.34±0.33	0.22±0.06	0.36±0.35	0.65±0.60	0.36±0.35	0.31±0.17	0.492	0.057
PCT*	–	0.09±0.12	0.12±0.16	0.08±0.11	–	–	–	–	–

2.5 Coinfection with other respiratory pathogens

2.5.1 Tuberculosis

COVID-19 patients have been linked to a respiratory infection, according to reports. The first of these is TB. Tuberculosis, both latent and active, is a risk factor for COVID-19 patients. Chen and colleagues presented their research. Interferon-gamma ray emitted by peripheral blood is used to diagnose tuberculosis in a patient. Patients with latent or active tuberculosis, as well as those who had been diagnosed with COVID-19 infection, were all suspects in this investigation. The symptoms in COVID-19 patients were more severe and occurred more quickly. [2] According to this study, corona virus patients should be checked for tuberculosis. Case reports later revealed that tuberculosis in coronavirus patients is diagnosed using the gene-Xpert MTB/RIF spectrum assay as well as a positive smear.



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Following that, a study was released that included 49 instances from eight countries across three continents. The author grouped the 49 examples into three groups:

1.Patients who had tuberculosis before having COVID-19.

2.Patients who had both COVID-19 and tuberculosis in the same week.

3.Patients with COVID-19 followed by tuberculosis.

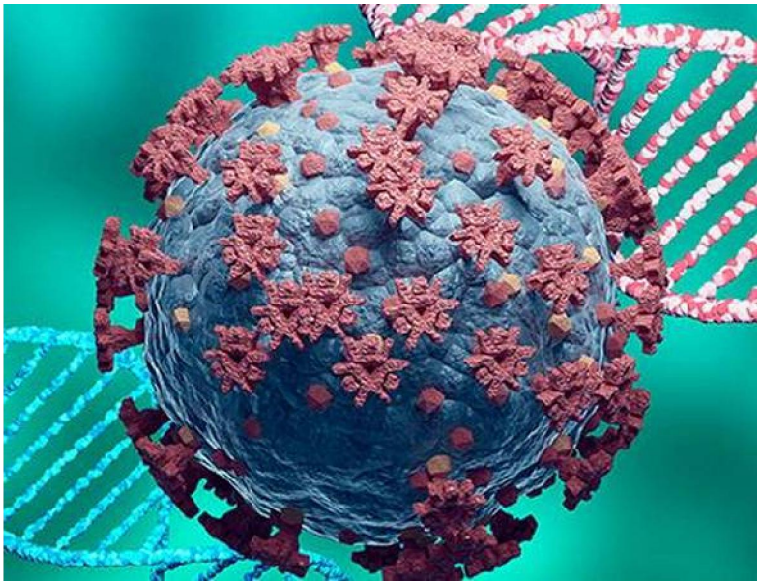
The result was reached after reviewing the literature on the suspension of the search and the time difference. Tuberculosis is a long-term sickness, whereas coronavirus is a one-time infection. The cause of co-infection was completely accidental. Nevertheless, there was concern about the high death rate associated with the research due to apparent Co-infection, which is about 12.3%, which is significantly higher than COVID-19 alone. [4] Co-infection was thought to be just coincidental in the above-mentioned comments. However, it is a severe problem in nations where TB rates are rising. This has a wide range of effects on the world's social and economic systems.

2.5.2 A typical pathogen

Several cases have been documented in which details about SARS-CoV-2 and Legionella have been described. It was a case of co-infection. Olivia and colleagues reported a large number of cases of SARS-CoV-2 and Mycoplasma/Chlamydia infection. Later in this piece, Nicholson and his colleagues examine the evidence for investigating the progression of COVID-19 disease and infection in order to determine its deadly outcomes.

2.5.3 Fungi

Articles have been published in respect with the presence of coronavirus disease in association with *pneumocystis jirovecii* pneumonia. In some patients suffering with coronavirus the fungal infection was seen simultaneously and, in some patients, the fungal infection was developed during the treatment of COVID-19. This type of phenomena was often seen in patients suffering from HIV disease.



2.5.4 Other

Several other infections were seen in association with SARS-CoV-2 namely measles and dengue. This was majorly seen in healthcare workers. This is a typical example for helminthic Co-infection. It can cause an increase in mortality and morbidity in corona cases due to less immunal response.

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2.6 Viral-Bacterial Interactions: Mechanism

We shall define the approaches related with microbial Co-infection and super infections in this section of the literature. We'll also observe how viral and bacterial infection work together. A lot of information about bacterial pathogens and influenza viruses, especially S.pneumoniae, has been gathered from the literature. We go over the mechanisms of these contacts, as well as the systematic interactions of SARS-CoV-2 with different pathogens, later in the literature. At the conclusion of the literature review, we put together a synopsis of the probable function of activated platelets and their immune response in increasing the risk of SARS-Cov-2 Superinfection and Co-infection.

2.6.1 Synergistic effects: Influenza virus and *S.pneumoniae*

Pneumococcus is the most common cause of secondary bacterial pneumonia, followed by influenza virus. This could result in a high risk of mortality. This was confirmed between the 1918 and 2009 pandemics. This study's mechanism includes the following:

i) It could cause a destruction in respiratory epithelium tissue.

ii) A rise in the concentration of chemicals that bacteria utilise as receptors could occur (This causes an increase in attachment of bacteria to the lung epithelium tissue).

iii) It can damage the function of immune cells such as macrophages and neutrophils, which are primarily responsible for the discharge of interferon-gamma during T-cell reaction to influenza. It can also affect pneumococci adhering to alveolar macrophages in the lungs.

The infection induced by influenza is depicted in the mechanism above. Pneumococcal colonisation, transmission, and active illness have all been aided by influenza.This is an independent upregulation of platelets activating receptor. A study was conducted in which mice were exposed to a non-lethal influenza virus. After seven days, the mice were completely dead. [2] Later, the mice were given a reduced form of the virus, which showed protection against influenza, and, in this case, mice survived. The substantial mortality caused by S.pneumoniae during the 1918 and 2009 pandemics has been reported in several other research.

2.6.2 Secondary bacterial infection associated with SARS-CoV-2: Molecular pathogenesis

Several articles on the mechanism of viral infection generated by SARS-CoV-2, which may lead to bacterial infection in the future, have been evaluated. The research focuses on the consequences of virus damage on adaptive and innate immunity in the respiratory epithelium. The most essential processes are that antagonising IFN responses increases microbial adhesion, colonisation, expansion, and causes a damage into healthy lung tracts. They then turn these mechanisms into the SARS-Cov-2 virus's recognised form.

Mirzaei and colleagues provided a synopsis for bacterial co-infection with viruses, particularly coronavirus. They proposed that bacterial co-infection causes SARS-CoV-2 infection. SARS-Cov-2, according to Manna and coworkers, is similar to SARS-Co-V, which was previously found to affect immune function-related gene expression in human monocytes.

According to a study on immune-related gene expression, SARS-CoV-2 de-regulates cathepsin or the proteasome, as well as IFN inducible. It synchronises cytokine receptors, chemokine receptors, MHC/ chaperon-related, lysosome-related, TLR/TLR signalling, and fibrosis-related genes in diverse ways. SARS-CoV is utilised to decrease type 1 IFN production, according to a study. It is the activity performed to compromise the alveolar macrophage recruitment and its functioning. Immune gene deregulation and differential regulation are mechanisms that generate a favourable environment for subsequent bacterial infection. It also promotes bacterial adhesion to host structural cells and activates the pro-inflammatory system, suppressing antibacterial host defence. Later, Bamford and Bogeochea raised a doubt on perturb gut homeostasis of SARS-CoV-2. The main function of the gut long axis is to prevent bacterial pneumonia, which can disrupt the gut microbiota. Its mechanism can have serious consequences in people who have been infected with the coronavirus, as well as secondary lung infections.Finally, Golta and colleagues discovered that human COVID-19 improves the adhesion of S. pneumoniae virus-infected cell lines. These cell lines are capable of fully transforming into primary human airway epithelial cell cultures. Surprisingly, this enhances platelet activating factor binding correlated expression. The bacterial-PAF-R interactions have evolved in this way.This mechanism is only slightly important.

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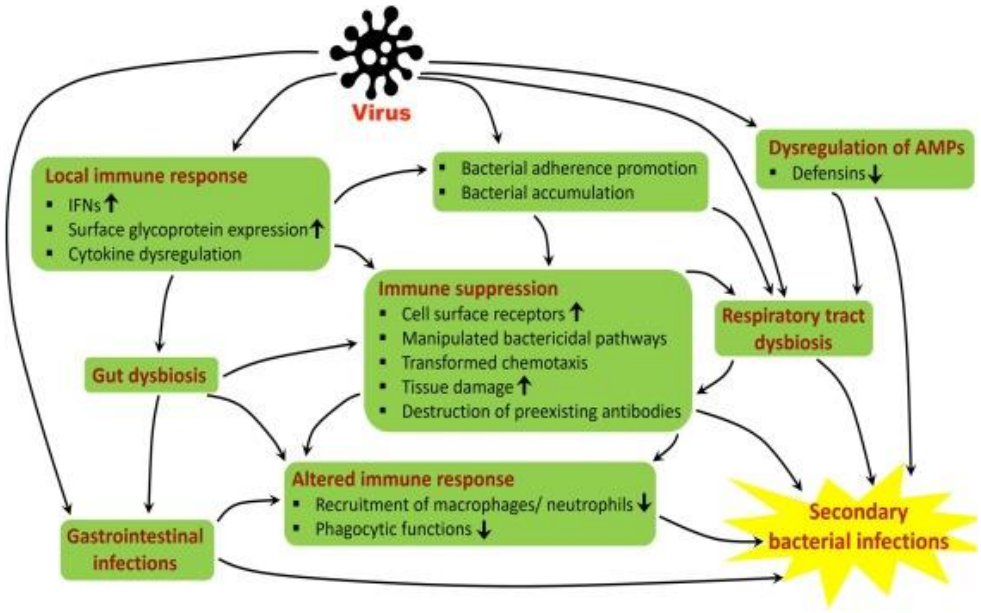
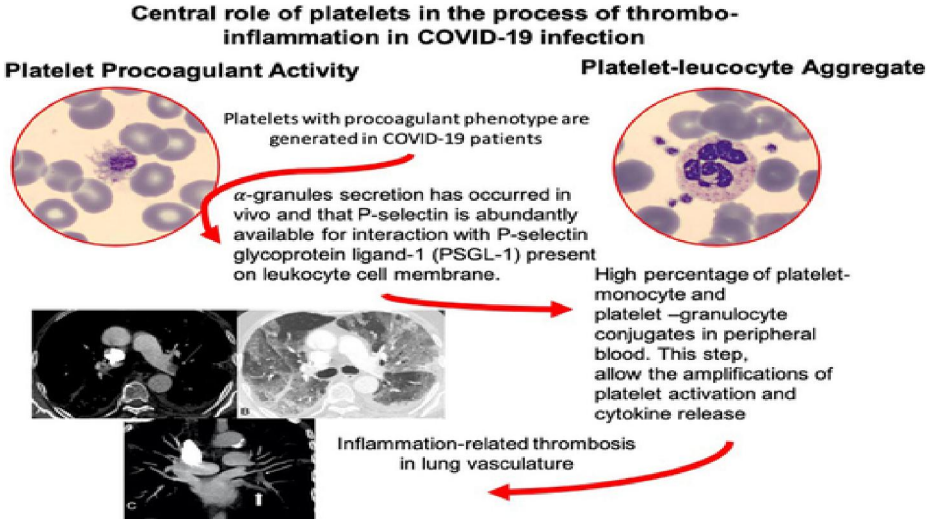


2.6.3 Pathogenesis: Platelets of SARS-Cov-2

Previous scientific examples and studies have highlighted the difficulty in discriminating between co-infection and super-infection after hospital admission of patients with severe COVID-19infection.Superinfection seems prominent in the SARS-COV-2 immunosuppression which is due to the existence of common disease-causing pathogen together with the poor defence mechanism or poor immunity to antimicrobial chemotherapy and unfavourable clinical consequences. This argument is strengthened by the threat posed by the inappropriate use of antibiotics in hospitalised patients with less severe disease, which contributes to the emergence of multidrug-resistant microbial pathogens in the hospital environment, posing a risk of nosocomial infection in patients who have been in the hospital for long periods of time due to severe coronavirus infection.



2.6.4 Platelets and NETosis

Platelets and associated inflammatory mediators such as reactive oxygen species, HMGB1, IL-8, and CD62P cause the formation of neutrophil extracellular traps (NETs). NETs play a key role in the development of SARS-CoV-2-related ARDs and cardiac injury. [10] The histone components of NETs, as well as other granule-derived proteinases, are

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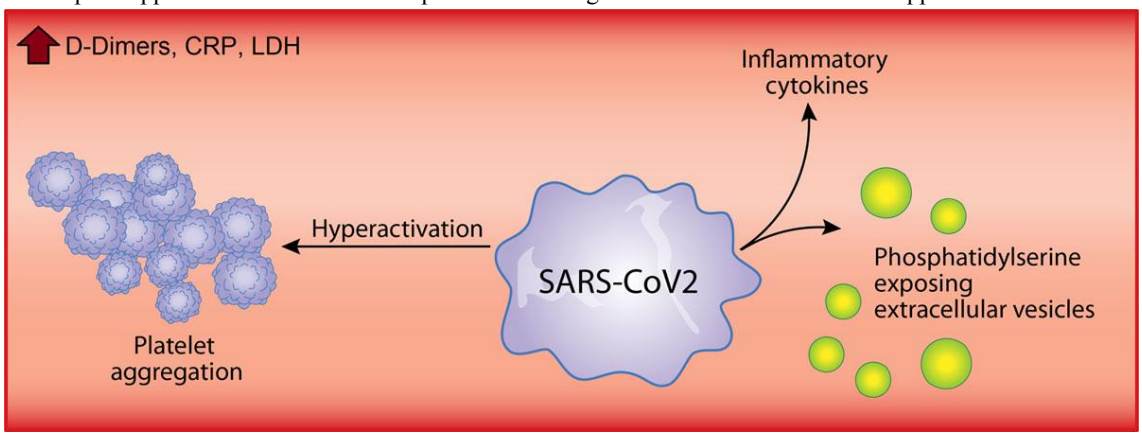
potent cytotoxins for epithelial and vascular endothelium, and they play a role in intravascular and intrapulmonary blockage.NET-derived histone- and proteinase-mediated cytotoxic effects on epithelial and endothelial cells following infection with the human coronavirus NL63, as well as SARS-CoV-2, the influenza virus, and other respiratory viruses are probable major contributors to the development of secondary and super-bacterial infections. This results from several mechanisms, including:

1. The exposing of receptors to bacterial adherence occurs when epithelial and endothelial cells are injured.
2. In epithelial and endothelial cells, bacterial adhesins unleash latent potentially dangerous intracellular pathogens.
3. Through the promotion of bacterial pathogen extrapulmonary dissemination.

Given the ambiguity around the involvement of the "cytokine storm" in COVID-19 pathophysiology, targeting platelets and neutrophils appears to be a viable technique for combating COVID-19-related immunosuppression.



2.6.5 Platelet-driven immunosuppression

COVID-19-related severe systemic inflammation is seen in human platelets and causes widespread immunosuppression, acute respiratory distress syndrome (ARDS), and heart failure, further complicating the illness. When the spike protein of SARS-CoV-2 interacts with angiotensin-converting enzyme 2 (ACE2) from megakaryocytes, it causes platelet activation and increases mean platelet volume and thrombocytopenia [9].

However, the presence of ACE2 mRNA and protein in platelets from COVID-19 patients is difficult to detect [10], implying the existence of alternate pathways of SARS-CoV-2 platelet activation. Although platelet hyperactivation causes immunosuppression and microvascular obstruction as a result of inflammation.

III. METHODOLOGY

The information for this article was acquired from a variety of sources, including BMC Bioinformatics, Antibiotic Discovery and Development, and others. Google Scholar, E-libraries, and Mendeley are examples of other online resources from where the data has been gathered. The report is based on a review of several research articles.

IV. RESULTS & DISCUSSION

COVID-19's present outbreak is life-threatening; according to studies, the version of COVID-19 infecting various other respiratory and organ problems in 2021 is different than the variant infecting various other respiratory and organ disorders in 2020. Recently, the WORLD HEALTH ORGANIZATION designated it as an air-borne disease that poses a greater risk. According to the World Health Organization, viral respiratory infections predispose people to bacterial infections, and this infection has the worst outcome of any infection. However, the precise significance of co-infection and super-infections in coronavirus patients is still unclear.

V. CONCLUSION

We can conclude from the above article that patients with Covid-19 infection are infected with a variety of infections. Co-infection or True infection can be used to describe certain pathogens. In patients who have the Corona virus, these infections can have serious consequences. Furthermore, determining which patients are suffering from Superinfection

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or Co-infection is quite challenging. Several recommendations have been given in regard to the efforts performed in determining the bacterial coinfection in current Covid-19 pneumonia patients. These suggestions are backed up by the facts presented in the preceding article.