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REVIEW ARTICLE

Bacterial co-infection in hospitalized COVID-19 patients

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ABSTRACT

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Background: Phototherapy is the most common way to help babies Coronavirus disease 2019; which is accountable for the contemporary pandemic, is a contagious illness produced by SARS-CoV-2 (severe acute respiratory syndrome coronavirus 2) which started with an outbreak of a febrile respiratory disease in China in December 2019. It is strongly believed that co-infections play an important role during COVID-19 outbreak. Furthermore, secondary bacterial pneumonia rates increase rapidly in intensive care unit patients. This study directed to elucidate the rate of bacterial contagions among hospitalized COVID-19 patients with documentation of the most common organisms to guarantee the responsible use of antibiotics among these patients. Our study's conclusions imply that ongoing observation of bacterial co-infection and resistance patterns, together with advancements in infection control techniques, are critical to managing the COVID-19 pandemic on a local and worldwide scale. Additionally, keeping track of the most prevalent microorganisms can aid in enhancing empirical antibiotic management guidelines for COVID-19 hospitalized patients, ensuring that antibiotic use is under control and excluding any potential side effects.

INTRODUCTION

For a long time, it has been recognized that viral respiratory contagions predispose to bacterial contagions [1]. Microbial super-infection during intensive care unit (ICU) admittance has been described in the other out-breaks of severe acute respiratory syndrome (SARS), but there is circumscribed data available concerning COVID-19 cases. Numerous authors identify the importance of super-infection but absolute data is still deficient [2] [3].

Consideration of co-infection in COVID-19:

One significant information gap that has been identified is the incidence and features of bacterial infection in patients infected with SARS-CoV-2 and severe acute respiratory syndrome [4]**.** It is strongly believed that co-infection performs an important role during COVID19. At the outset, COPD is an important comorbid condition correlated to critical COVID-19 [5]**.** Even in the stable phase of COPD patients, bacterial pathogens remain present, increasing the likelihood that SARSCoV-2 infection will arise in patients who already have bacterial infection. Second, super-infections by nosocomial antibiotic-resistant bacteria may be the cause of the rise in secondary bacterial pneumonia rates among patients admitted to the intensive care unit. Hospitalization is necessary for critically ill COVID-19 patients, which raises their risk of developing secondary bacterial pneumonia. Recent research indicates that 3.13.5% of COVID-19 patients had bacterial co-infection at admission, and up to 15% of patients had additional bacterial infections after being admitted to the hospital [6]**.** Patients with COVID-19 typically stay in the hospital for seven days, but this can go up to fourteen days or more. The risk of developing hospital acquired pneumonia (HAP) rises significantly with extended hospital stays. Moreover, mechanical ventilation (which is utilized to treat COVID-19 patients who have been hospitalized to the intensive care unit) is linked to more than 90% of HAP cases. Reports of bacterial co-infections in MERS-CoV patients admitted to the intensive care unit have also surfaced [7]**.**

Antimicrobial stewardship

COVID-19 cannot be effectively treated with antibiotics. On the other hand, a number of recommendations support the use of empirical antibiotics in patients with severe COVID-19, whether the infection is suspected or confirmed [8]**.** These suggestions, however, bring up issues with the overuse of antibiotics and the damage that results from bacterial resistance. It's critical to determine whether co-infections occur in COVID-19 patients and whether this justifies the necessity of initial empirical antibiotic treatment. This is because there is concern about the potential complications and side effects of antibiotic abuse, which could lead to the development of resistant hospital acquired microorganisms and undermine the principles of antimicrobial stewardship programs [9]**.** Therefore, in order to treat COVID-19 patients, assure appropriate antibiotic administration, and reduce the harmful effects of overuse, it is vital to determine the percentage of patients with secondary bacterial pneumonia and the species that cause it [4].

General features of viral infection in case of Co-Infection:

It is known that viral respiratory illnesses, both upper and lower, can progress differently. Some illnesses may be largely asymptomatic, and symptoms might vary from a few days to several weeks. With rare exceptions, the presence of live virus during infection usually disappears after 10 days. Long-lasting disease of the afflicted airways of varying intensity may accompany more severe respiratory illnesses. Co-infection is possible either during the early phases of acute viral excretion or after clinical disease residues become apparent. Comorbid conditions may also influence the result [10]**.**

Effect of bacterial co-infection during COVID-19:

Three unlike bacterial/SARS-CoV-2 co-infection conditions can be imagined:

1. SARS-CoV-2 that resurfaces after bacterial infection.
2. Bacterial and viral pneumonia together.
3. Following SARS-CoV-2, a secondary bacterial super-infection.

The mechanisms underlying these circumstances, which involve composite interactions between the virus, host, and bacteria, are highly context- and time-dependent. Although a mixed viral and bacterial pneumonia most likely does not elicit the same immune response to SARS-CoV-2. Co-infections with bacteria can reduce host defensive signals, which increases vulnerability to SARS-CoV-2 infection and the ensuing pathology. For instance, respiratory infections, such as Klebsiella pneumoniae, restrict the host antiviral program's ability to activate NF-kB-governed responses [11].

COVID-19 secondary bacterial infection molecular mechanisms:

Researchers have revealed that viruses injure the respiratory epithelium and affect both innate and adaptive immunity, which in turn counteracts IFN responses that promote bacterial adhesion, colonization, growth, and invasion into healthy respiratory tract sites. These are significant mechanisms [9]. An insightful analysis of bacterial co-infections with viruses, generally, and SARS-CoV-2, particularly, is presented [12] examining in depth potential hypothesized mechanisms of virus susceptibility to bacterial co-infection, simultaneously postulating the processes by which SARS-CoV-2 bacterial co-infection happens and offering practical recommendations for their management and prevention. Manna et al. then reported that SARS-CoV-2 is like SARS-CoV, which has been reported to regulate gene expression linked to immune function in human monocytes [13].

Role of platelets in SARS-CoV-2 secondary infections:

The role of platelets in causing the excessive systemic inflammation associated with COVID-19 is becoming more and more well-documented. This inflammation not only leads to widespread immunosuppression but also complicates acute viral disease by causing ARDS and cardiac failure [13]. It has been found that ACE2 is expressed by platelets, which are most likely derived from megakaryocytes. ACE2 activates platelets via interacting with the S-spike protein of SARSCoV-2. Indicators of platelet activation caused by this process include the up-regulation of adhesion molecule expression, cellular aggregation, CD62P, activation of the integrin, GP11b/111a, mobilization of α-granule and dense granules, and platelet spreading. [14]. Proinflammatory activities of platelets found in vitro are linked with thrombocytopenia and increased mean platelet volume. Others, however, were unable to detect whether the protein or the mRNA encoding ACE2 was present in platelets from COVID-19 patients [13].

Clinical statistics on co-infections with COVID

It was discovered that 5 (10.6%) of the SARS-CoV-2 infected patients who require mechanical ventilation for acute respiratory distress syndrome also had multiple pathogen co-infection, while 13 (27.7%) patients had early bacterial organism co-infection. Another Spanish study of ICU patients identified both early and late infections in 92 patients. Generally, out of 24 patients (26%), 32 microbiological isolates (mainly Staph. aureus, Streptococcus pneumoniae, and H. influenzae) were found within 48 hours. Although their hospital stays prior to ICU admission were longer (median 9 days) than those of the general group (median 3 days), Pseudomonas aeruginosa was detected in several of these individuals [15].

CONCLUSION:

Our study revealed that Critically ill COVID-19 cases exhibited high rates of bacterial and fungal co-infections in their sputum (80.8%), which was strongly associated with poor outcomes. This suggests that antibiotic use may be an essential part of the therapeutic arsenal for some patients with severe COVID-19.

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