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Management and Prevention of Bacterial Pneumonia and Respiratory Co-Infections in COVID-19 Patients

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Introduction

A Polymerase Chain Reaction (PCR) assay should be used to test all patients with respiratory infection symptoms for influenza, in addition to SARS-CoV-2. If different respiratory viruses are accessible, PCR assays can be used. All patients with influenza A or B viral co-infection should be treated with oseltamivir or an alternate drug, regardless of disease severity. If there is an evident exposure or risk factor, empiric treatment for influenza virus co-infection can be explored while waiting for test results. Treatment options are limited and successful only in specific conditions such as immunosuppression or hypogammaglobulinemia if viral co-infection with another respiratory virus, such as respiratory syncytial virus, is discovered. In order to establish the benefits of such treatment in light of the potential risk of aggravating COVID-19 related organ failure and the potential side effects of the medicine or medications, infectious disease consultation is strongly suggested.

Bacterial Pneumonia

COVID-19 requires a strong index of suspicion to diagnose combined viral and bacterial pneumonia or subsequent bacterial pneumonia. Despite a large overlap of viral and bacterial symptomatology, several signs of bacterial infection may still be discernible. Neutrophil leukocytosis is a hallmark of bacterial pneumonia, but COVID-19 patients frequently present with lymphopenia and a normal white blood cell count. When it comes to determining the cause of community-acquired pneumonia, procalcitonin is neither sensitive nor specific. On the other hand, several studies of COVID-19 cases have repeatedly showed normal (low) procalcitonin levels in isolated SARS-CoV-2 infection, leading to its widespread albeit invalidated use to rule out combination viral and bacterial pneumonia, albeit the exact threshold remains unknown. In the context of a therapeutic setting, this remark emphasizes the significance of taking into account all variables. The risk of mixed viral and bacterial pneumonia is low in patients with mild to moderate respiratory failure consistent with COVID-19 and no clear indications of bacterial infection, thus antibiotics can be safely avoided. The advancement of COVID-19 in this instance is more likely than a new superimposed secondary bacterial pneumonia to cause gradually increasing respiratory failure during the first week of presentation. Patients who begin with noninvasive forms of supplementary oxygen and eventually require invasive mechanical breathing fall into this category.

Antibiotics should not be started unless there is evidence of bacterial pneumonia, even if respiratory discomfort is worsening. However, unless proven otherwise, if a patient develops new or acutely worsening respiratory failure, sepsis, or both after an initial period of consistent improvement (considered to be days), then nosocomial acquisition of secondary bacterial infection, *i.e*, secondary bacterial pneumonia in the form of hospital-acquired pneumonia, infection at an extra pulmonary site or both is likely. While COVID-19 can produce acute respiratory decompensating on its own, there is little information on the role of subsequent bacterial pneumonia in such decompensating. As a result, empiric antibiotic therapy based on guidelines may be appropriate until the secondary infection is ruled out. New or recrudescent fever; new onset or change in the character of sputum; new leukocytosis or neutrophil (or both); new relevant imaging abnormalities; and new or increasing oxygen demand are all examples of supporting evidence for secondary bacterial pneumonia. In these patients, any alternative sources of hospital-acquired infections, such as indwelling central venous catheters or urinary tract catheters, must be considered and treated appropriately.

Bilateral Infiltrates

A severely ill patient admitted with acute respiratory failure requires immediate empiric treatment for all suspected reasons. This is especially significant since procalcitonin levels in individuals with multiorgan failure might be mistakenly increased, and imaging investigations can be difficult to distinguish between bilateral infiltrates of acute respiratory distress syndrome and concealed consolidation of bacterial infection. The guidelines of the Infectious Diseases Society of America (IDSA) and the American Thoracic Society (ATS), as well as host risk factors and past microbiologic data, should be used to guide empiric therapy for community-acquired pneumonia. All patients should have respiratory samples tracheal aspirate is preferred to sputum in mechanically ventilated patients and blood cultures sent, ideally before antibiotics are started. All patients with severe community-acquired pneumonia should have their urine antigen tested for Streptococcus pneumonia. Based on the clinical setting and epidemiology, Legionella pneumophila urine antigen and Mycoplasma pneumonia IgM and IgG antibodies can be provided.

A positive respiratory culture can indicate colonization even if there are no indications of bacterial pneumonia, especially in patients who have had previous pneumonia with the same organism or have altered airway anatomy. This differentiation can be made using laboratory markers, radiologic characteristics, and quantitative and semi quantitative culture approaches. Secondary bacterial pneumonia in a patient on invasive mechanical ventilation presents similarly to hospital-acquired pneumonia, but it necessitates the use of empiric broad-spectrum antibiotics that cover mrsa, Pseudomonas aeruginosa, and possibly other multidrug-resistant organisms, as per the guidelines. Antibiotic side effects and institutional antibiograms must also be taken into account. Patients with ventilator-associated tracheobronchitis generally do not show the conventional indications of secondary bacterial pneumonia have increased secretions and low-grade fevers and are difficult to wean off ventilator assistance. Antibacterial therapy for this clinical entity has limited evidence to

support it, necessitating a careful case-by-case examination. In the absence of problems, antibiotic therapy lasts 5 days to 7 days for community acquired pneumonia32 and 7 days for hospital-acquired pneumonia and ventilator associated pneumonia19. If patients show symptoms of clinical stability, especially if unfavorable effects are observed, consider decreasing the period. Checking the procalcitonin level upon presentation can aid with antibiotic de-escalation depending on the procalcitonin level pattern over the next 24 hours to 48 hours. It is permissible to stop all antibiotics if a microbiological source is not found within 48 hours of testing and the procalcitonin level is less than 0.5 g/L or declines by 80 percent or more from peak concentration.

Because they suppress frequent indications of sepsis, the use of Interleukin 6(IL-6) inhibitors such tocilizumab for COVID-19 related cytokine activation syndrome provides a distinct difficulty. Tocilizumab therapy for rheumatologic illnesses has repeatedly been linked to an increased risk of severe bacterial infections. C-reactive protein and other acute-phase reactants, such as white blood cell count, can be unreliable and may not rise in response to a secondary bacterial infection following tocilizumab treatment. It's unknown how long this impact lasts with just one or two doses. IL-6 inhibitors may have less of an effect on procalcitonin, however the data to distinguish bacteria from viral pneumonia in this situation is limited and should be investigated further. Finally, invasive pulmonary aspergillosis has been reported in critically ill patients with seasonal and pandemic influenza, and is linked to a high rate of morbidity and mortality. Patients with COVID-19–associated acute respiratory distress syndrome were also found to have invasive pulmonary aspergillosis. Patients with immune-compromising diseases, prior or simultaneous influenza viral co-infection, clinical deterioration despite appropriate antibiotics, and positive fungal indicators such as galactomannan on culture should be examined for this complication. If invasive pulmonary aspergillosis is suspected, treatment with a wide antifungal such as voriconazole should be started as soon as possible after consulting with infectious disease specialists.