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Short Communication

Physicians Take Heed, the Utilization of Dexamethasone and Broad Spectrum Antibacterial Antibiotics in COVID-19 Therapy Carries with it the Risk of Secondary Bacterial and Fungal Opportunistic Infections

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Quotation: "The physicians of 2020 who are treating coronavirus COVID-19 patients must take heed and never forget the lessons of the past involving the risks associated with the use of glucocorticoids (Dexamethasone), and the use of broad spectrum anti-bacterial antibiotics, as relates the risk of secondary bacterial and fungal opportunistic pathogens".

Coronavirus COVID-19 is associated with an extremely infectious respiratory droplet infection that is spread from person to person by coughing, sneezing and aerosols general by oral speech [1,2]. This virus was first isolated and identified in Wuhan, China in 2019 [3].

This viral infection is associated most often with such symptoms as: fever, dry cough, fatigue, and shortness of breath [4,5]. Symptoms of this disease make their appearance around 2 - 14 days from the time of exposure [1-5]. The vast majority of cases of COVID-19 associated with rather mild symptoms but, this viral infection can evolve into pneumonia which can then spread even further to involve multiple organ failure ultimately leading to a fatal outcome [1-5]. Reports have cited the occurrence of additional neurological manifestations due to COVID-19 [4-9].

Horby., *et al*. cited the fact that COVID-19 infections are often linked with the appearance of diffuse lung damage [10]. These investigators stated that perhaps Glucorticoids could serve to "modulate inflammation-mediated lung injury" [10]. These investigators further noted that by modulating inflammation-mediated lung injury, glucocorticoids could thus reduce "progression to respiratory failure and death" [10].

Horby., *et al*. found that in their patients hospitalized with Coronavirus COVID-19, the application of dexamethasone (glucocorticoid) brought about over a twenty-eight day period of time a lower death rate in those patients who received: a) invasive mechanical ventilation or b) were given oxygen alone. Dexamethasone did not bring about a lower rate of mortality in those patients who were not given respiratory support [10].

The research investigation carried out by a team at Oxford University further substantiated the earlier findings Horby., *et al*.. , and The Oxford group reported that dexamethasone should be utilized without delay as a standard treatment regimen in COVID-19 patients with respiratory distress [11].

Chen., *et al*. found that glucocorticoids work by acting upon the intracellular glucocorticoid receptors [12]. These investigators thus found that as concerns immune system, the glucocorticoids could affect both "innate and adaptive immunity" [12].

The study of Chen., *et al*. looked at immunoregulatory effects of dexamethasone treatment on mouse splenic T, Treg NK and NKT cells. Dexamethasone treatment was found by Chen., *et al*. to decrease number NK cells and also to suppress their activity [12]. These investigators concluded that Dexamethasone "may suppress T and NK- mediated immunity" [12].

Sewell *et al*, noted that immunity is both protective and enduring vis a vis infections involving viruses as relates to the combined actions of B lymphocytes (humoral immunity) and T lymphocytes (cellular immunity) [13]. These investigators further noted that in order to have an effective immune response to SARS-COV-2 which includes the involvement of four types of T-cells [13].

It is the T helper cells (CD4) that are responsible for cellular immunity and which also aid the B cells to produce neutralizing antibodies [12,13].

The cytotoxic T cells that are also called killer T cells (CD8) that kill infected cells. Additional T cells are linked to the inflammatory reactions that help control infections. The final specie of T cell called the regulator T cells (T regs) helps with the containment of the immune response and thus serves to prevent over-reaction that results in tissue damage [12,13].

Ni., *et al*. examined blood samples from patients convalescing from COVID-19 infection and detected SARS-CoV-2 specific humoral and cellular immunity [14]. Ni., *et al*. also found that most of their patients manifested neutralizing activity in their serum which they also found, correlated with "numbers of virus specific T cells" [14].

Lionakis., *et al*. noted that since the early 1990's the presence of opportunistic infections served as an important cause of morbidity and mortality in immunocompromised patients [15].

Zhou., *et al*. have noted that both bacterial and fungal infections have been found to be common agents of secondary infection in viral patients, especially those individuals who are critically ill [16]. Zhou also noted that in SARS patients, gram negative bacilli were most commonly encountered bacterial agents of infection, *Candida species were* the most frequently encountered agents of mycotic infection [16].

Sadly Zhou., *et al*. stated that bacterial and fungal opportunistic infections have not been adequately studied in COVID-19 patients [16]. What is even more shocking was that Zhou., *et al*. mentioned that the currently utilized infection control protocols which have been used to date, have focused on the preventing transmission and cross infection by SARS-Co-V-2, Physicians however, have totally failed to address the issue of preventing bacterial and/or fungal secondary/opportunistic infections in critically ill Coronavirus COVID-19 patients [16].

Zhou., *et al*. goes even further to mention that in China there has been little attention paid to address the issue of secondary bacterial an fungal infections, especially as relates to the creation of a standardized diagnostic process [16]. These investigators note that it is important to have clinical data as relates to secondary bacterial and fungal infections because this data is valuable for "guiding evidence based treatment" for COVID-19 [16].

Bedard has also noted a new and dangerous development in the evolving story of coronavirus COVID-19 pathology, namely, that patients are at increased risk of acquiring fungal Infections [17].

Bedard points out that pulmonary Aspergillosis (CAPA) was the actual cause of pneumonia in COVID-19 patients admitted to the hospital [17].

Bedard also echoes the earlier finding of Zhou, namely, that fungal infections in COVID-19 patients comprised a very serious threat that has been largely ignored by medical authorities [16,17].

Bedard further remarked that among the fungal species isolated in COVID-19 Patients, 39% were *Aspergillus* species which were isolated from tracheal aspirates. In this group of patients, 21.7% had Pulmonary Aspergillosis. In this same group of patients, 17.4% also had *Aspergillus* colonization which was associated with a fatality rate of 44% [17].

Bedard stated that an important reason why there have been increases in the presence of secondary fungal infections besides immune dysfunction has to do with the fact that patients were being given broad spectrum antibacterial antibiotics, parental nutrition, and invasive examinations [17]. All of these aforementioned facts can bring about the risk of increased cases of *Candida* infection [17]. Bedard also notes that COVID-19 patients exhibited an over expression of inflammatory cytokines, impaired cell mediated immunity [17].

During the early days of the AIDS Epidemic in the late 1980's, impaired cellular immunity had been linked to the increased appearance of opportunistic secondary fungal infections [18]. Glucocorticoids (Dexamethasone) use can adversely affect cell mediated immunity, thus favoring the appearance of fungal pathogens [11.12,18,19].

Louria had noted back in 1974 that the administration of anti-bacterial antibiotics promoted systemic yeast infections [19]. The yeast infections most commonly found were those associated with the Genus *Candida* [19]. Louria further stated that the "sequence of pathogenesis" involved: 1) suppression of normal bacterial flora, and 2) colonization with *Candida* [19].

Thus, the physicians of 2020 who are treating coronavirus COVID-19 patients must take heed and never forget the lessons of the past involving the risks associated with the application of glucocorticoids (dexamethasone), and the use of broad spectrum anti-bacterial antibiotics, as relates the risk of acquiring secondary bacterial and fungal opportunistic infections [11,12,18,19].

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