Journal of Infectious Diseases and Research
JIDR, 4(S2): 15
www.scitcentral.com
ISSN: 2688-6537

Abstract: Open Access

Data from Trimethoprim and Cotrimoxazole in Severe Covid-19 Lung Injury

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Published October 13, 2021

ABSTRACT

The commonest cause of mortality in Covid-19 is acute lung injury. Post mortem studies show high levels of stressed neutrophils in the alveolar capillary bed extruding their nuclear material as neutrophil extracellular traps (NETosis); these are pro-thrombotic and can block the alveolar capillary bed causing profound hypoxia and death. Neutrophils are stressed by oxygen free radicals (ROS) generated by a cytokine storm. Released formyl peptides from tissue injury will cause homing of neutrophils to the lung. The predominant mechanism of neutrophil activation is via their surface formyl peptide receptors (FPR's) responding to infection or inflammation. Over stimulation of the FPR by cytokines drives the neutrophil to also release extracellular ROS. This feeds back onto their FPR's and if intense will drive NETosis. NETosis aims to trap infectious particles. Both cotrimoxazole (CTX) and trimethoprim (TMP) block the surface FPR's reducing neutrophil traffic to the lung and NETosis. Several case studies of these drugs in Covid-19 show reduced mortality and reduced length of stay (LOS). Improvements were seen within 48 h. A TMP case series (n=44) showed mortality to be 4.5% with added TMP versus 32% without. LOS was also reduced (9 days versus 22). India data for critical Covid-19 (n=201) showed mortality was 13% with added CTX versus 40% without; the need for ventilation was reduced by 27%. LOS was reduced (11 days versus 15). 14 patients with severe Covid-19 treated at home with oxygen and CTX, showed zero mortality with only one admitted for enteric fever. Further UK data is being analyzed for TMP and a randomized study of CTX in critical Covid is ongoing in India. Both drugs are inexpensive and licensed for respiratory illness and data suggests that they show benefit in oxygen dependent patients with acute lung injury.

Keywords: Covid-19, Acute lung injury, Trimethoprim, Cotrimoxazole, Mortality

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Citation: Quadery SR & Varney VA. (2021) Data from Trimethoprim and Cotrimoxazole in Severe Covid-19 Lung Injury. J Infect Dis Res, 4(S2): 15.