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Theory and Practice of Successful Treatment of COVID-19 with High-Dose Colchicine

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About the study

It is well known that around 80% of COVID-19 patients recover without specific treatment, 15% develop "severe" COVID-19 and 5% end up with "critical" COVID-19 requiring special assistance at critical care management units. The main cause of mortality is the Cytokine Storm (CS), leading to the severe symptoms of Acute Respiratory Distress Syndrome (ARDS), hemodynamic instability, disseminated intravascular coagulation or multiple organ failure. The cause of CS is the hyper reaction of the Nod-Like Receptor Family, Pyrin Domain Containing 3 (NLRP3) inflammasome. Myeloid cells are the major source of dysregulated inflammation in COVID-19 thus hyper activation of the NLRP3 inflammasome and the subsequent CS take place precisely inside them [1]. Colchicine's distinguishing feature is its remarkable ability to accumulate intensively in leukocytes, where the CS is generated. Whereas the peak plasma concentration after a single oral dosing of 0.6 colchicine is approximately 3 nmol/L, a saturable accumulation of 40 nmol/L to 200 nmol/L of the drug has been shown to occur in neutrophils. Thus, it is logical to expect that by raising the doses of colchicine within acceptable limits, a level of its concentration sufficient to inhibit the NLRP3 inflammasome in leukocytes can be reached. Increasing doses of colchicine can lead to such an accumulation in macrophages, neutrophils and monocytes that is sufficient to inhibit the NLRP3 inflammasome and, accordingly, the CS.

Practice

In March 2020 we started the administration of higher doses of colchicine due to the fact that we did not get satisfactory results with the low doses of the drug. Our assumption was that safe increase in colchicine doses to reach micro molar concentrations in leukocytes will result in NLRP3 inflammasome inhibition [2].

The inhibitory effect of colchicine on CS occurs at doses, such as (0.5 mg per 10 kg body weight) -0.5 mg, but not more than 5 mg loading dose. This makes 0.04 mg/colchicine/kg-0.045 mg/colchicine/kg. We consider that doses of colchicine below 0.1 mg/kg are completely safe, and those between 0.1 mg/kg and 0.2 mg/kg may in some cases lead to toxicity side effects, but certainly not to death.

In 785 in patients treated with increasing doses of colchicine, mortality fell between 2 and 7 times. Our data, including also a large number of COVID-19 outpatients, showed that nearly 100% of the patients treated with this therapeutic regimen escaped hospitalization. In addition, post-COVID symptoms in those treated with colchicine were significantly less.

We have published series of cases demonstrating the life-saving effect of colchicine [3,4].

For example, a patient with only about 10% of the lung not being affected by severe bilateral pneumonia and ARDS, and obese patients with a BMI over 40 (and comorbidity) representing the most at-risk group for COVID-19 complications, are typical cases of the life-saving effect of high doses of colchicine in high-risk COVID-19 patients. It is interesting to note that accidentally taken single overdose of colchicine (15 mg or 12.5 mg) was sufficient for the complete recovery of patients, including the eradication of pericardial effusion.

The life-saving effect of high-dose colchicine was vividly demonstrated in the 101-year-old patient fighting for his life after two surgical interventions. While recovering in the intensive care unit, he was infected with COVID-19, but thanks to immediate treatment with high doses of colchicine, the patient was saved.

The failure of antivirals (paxlovid, molnupiravir, and remdesivir) to solve the problem of treating COVID-19 is due to the fact that there is no direct link between viral replication and the hyper responsiveness of the NLRP3 inflammasome. High doses of colchicine inhibit the NLRP3 inflammasome/CS and solve this problem.

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