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Commentary

Macrophage Activation in Severe Acute Respiratory Syndrome

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DESCRIPTION

A white blood cell called monocytes arises from the bone marrow. An innate immune response component called a monocyte controls cellular homeostasis, particularly in conditions of infection and inflammation. They make up about 5% of the nucleated cells that circulate in healthy adult blood. Circulating monocytes have a half-life of one to three days. Myelodysplastic syndromes frequently exhibit monocytopenia, which is a reduction in the number of circulating monocytes. While monocytosis, an increase in circulating monocytes, is a frequent finding in peripheral blood, particularly in connection with infection, trauma, medicines, autoimmune illness, and some malignancies, it is also seen in combination with other conditions. Investigation into the diagnosis of chronic myelomonocytic leukaemia is warranted when monocytosis is persistent and unexplained.

Activation of macrophages

With other highly virulent coronavirus infections, such as SARS-CoV and MERS-CoV, a similar illness course to that seen during SARS-CoV-2 infection has been described, with 20% of patients ultimately progressing to fatal ARDS. In the post-mortem lungs of these patients, extensive cellular infiltration dominated by macrophages was discovered. High amounts of Interferon (IFN), IL-6, IL-12, Transforming Growth Factor (TGF), CCL2, CXCL10, CXCL9, and IL-8 were found in patients with SARS-CoV, which is similar to what has been described in SARS-CoV-2. Low levels of IL-10 and high levels of IL-1 were frequently seen in SARS-CoV, in contrast to those infected with SARS-CoV-2. Notably, MERS-CoV can replicate in monocytes, macrophages, and dendritic cells while MNP infection in SARS-CoV fails.

It is unclear what causes highly pathogenic human coronavirus infections to cause severe lung disease. High viral replication rates may be the cause of increased host cell cytolysis and the robust production of inflammatory cytokines and chemokines by infected epithelial cells. Additionally, virus escape mechanisms, such as the production of interferon inhibitory proteins, may delay the induction of antiviral interferon responses, prolonging viral damage and causing an excessive buildup of monocytes, macrophages, and neutrophils in the body. Although viruses have developed numerous ways to inhibit the production of type I interferon in infected cells35, it is surprising that reduced interferon activity can endure in COVID-19 patients who experience severe inflammatory reactions7, this suggests that additional pathways, such as inflammasome activation, may be responsible for the ongoing low level of type I interferon induction, as was recently demonstrated36. It will be crucial to pinpoint the precise processes causing the decreased type I interferon activity in order to create specific immunomodulatory treatments for COVID-19 patients.

Studies in animal models of virus-induced Acute Lung Injury (ALI) reveal that a number of mechanisms, including the infiltration of monocytes, are responsible for pro-inflammatory programmes. For instance, type I interferon, oxidative stress, anti-spike protein IgG immune complexes, NLRP3 inflammasome activation, and other factors may all contribute to the sustained activation of invading monocytes and monocyte-derived macrophages. Interestingly, deletion of the Toll-Like Receptor (TLR)/IL-1 receptor adapter MYD88 had no impact on mouse ALI38; in fact, MYD88 was even shown to play a protective role, however this protection was not dependent on IL-1 receptor interaction by IL-143.

CONCLUSION

The innate immune system depends heavily on monocytes. They are the source of numerous additional crucial immune system components, including dendritic cells and macrophages. Monocytes are involved in both the pro-inflammatory and anti-inflammatory immune response pathways. Numerous hematologic illnesses, as well as inflammatory and immunological disorders, can be diagnosed using information on the presence or absence of monocytes.

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