Early alterations of blood monocyte subset distribution and surface phenotype are linked to infection severity in hospitalized COVID-19 patients

Manuscript file

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# Abstract

Severe SARS-CoV-2 infection manifests with systemic immediate pro-inflammatory innate immune activation and altered iron turnover. As iron homeostasis and monocyte differentiation and function are interconnected we sought to characterize how those two processes impact on the cellularity, surface marker expression and iron transporter phenotype of neutrophils and monocyte subsets in COVID-19 patients within 72 hours from hospital admission, and how these changes relate to infection severity. Between March and November 2020, a total of 48 blood leukocyte samples from COVID-19 hospitalized patients and 7 from healthy individuals were analyzed by flow cytometry enabling comparative analysis of 41 features. Apart from inflammation-driven neutrophil expansion, we corroborate the depletion of the CD16+ non-classical monocyte subset in hospitalized patients independent of the subsequent course of infection. Increased CD64 expression in neutrophils and monocytes as well as monocytic CD40 surface levels could be associated with disease severity. Interestingly, surface CD86 expression in the classical and intermediate monocyte subsets, which peaks in moderate COVID-19, was associated with a favorable course of the disease possibly indicating an effective antiviral response. We also found that low transferrin receptor expression on monocytes upon admission was associated with a poor course of COVID-19. These early alterations of the myeloid leukocyte compartment may be hallmarks of inefficient viral control and help in early risk prediction and to optimize treatment.

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