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Is CD16 Enough Marker to Define Circulating Monocytes Role?

Claudio Karsulovic[1,2]

[1]Internal Medicine Department, Clinica Alemana de Santiago.

[2]Laboratorio de Inmunomodulación Neuroendocrina, Instituto de Ciencias Biomedicas, Universidad de Chile.

***Corresponding Author:** Dr. Claudio Karsulovic M.D, Ph.D., Internal Medicine Department, Clinica Alemana de Santiago.*

To the Editor

During pandemic times monocytes have become important since are precursors of activated macrophages which are being related to alveolar damage seen on SARS-Cov2 patients(1). Before its differentiation to macrophage on the target tissue, circulating monocytes have phenotypes commonly associated with relative quantities of CD14 and CD16 on its membrane. In 2010, the International Union of Immunological Societies and the World Health Organization set the subset classification for circulating monocytes. CD14 (Monocyte identifying Toll-Like Receptor) and CD16 (FcyRIII co-receptor, a marker of inflammatory monocyte) were used to establish three subpopulations of monocytes with different features in terms of inflammatory protein expression and phagocytic capabilities(2).

For many years numerous studies have shown a correlation between a specific phenotype and a disease or group of them(3). Inflammatory diseases seem to correlate better to inflammatory phenotypes having higher CD16 on its membrane(4). On the other hand, neoplastic or more chronic inflammatory diseases appear to have increased CD14 and lower levels of CD16(5). Nevertheless, these observations are wildly accepted and in general reproducible, while new markers are added or different stages of diseases are analyzed individually, more complicated becomes its analysis.

In the context of SARS-Cov2 studies, circulating monocytes have been assess using conventional markers in serum samples instead of membrane attached ones. Recently, soluble CD14 has been described as increased in SARS-Cov2 patients, supposing that this elevation of serum values is capable of detect changes in monocyte activation(6). It is well known that the proportion of monocytes do not change after stimulation or pathological conditions(1, 3). This approach has serious issues since there is no consensus about how monocytes behave in terms of markers expression, days before tissue differentiation; and whether the rise in a serum marker is a reflection of the predominant inflammatory or non-inflammatory monocyte status.

To be able to prudently analyze more circulating monocyte status and correlate it reasonably to a specific disease and inflammatory status, it is imperative to clarify three main points first: one, is it enough two-markers based classification to attribute inflammatory characteristic to circulating monocytes?; two, if it is so, how do we determine an inflammatory or non-inflammatory monocyte?; three, if we are not able up to date to tell if monocytes shed their markers from the surface, how can we be sure that circulating monocytes are producing that serum marker measured?

Simple and easy experiments, exposing healthy and recently extracted monocytes to inflammatory and non-inflammatory stimuli will be needed to have a solid base to clarifying these three questions in the future.

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