Letters to the Editor

Age-related macular degeneration, apnea and macrophages

Degeneración macular asociada a la edad, apnea y macrófagos

Dear Sir,

Age-related macular degeneration (ARMD) is a multiple etiology disease in which a broad range of genes and proteins play significant roles. Wet or exudative ARMD, also known as neovascular ARMD, is the type which causes most loss of vision. Neovascularization is an active and inflammatory condition (cytokins, growth factors and inflammatory cells) in which the initial stage could be the interaction between M2 macrophages and the pigment epithelium. Multiple risk factors have been associated to ARMD, including sleep apnea (SA) (which is independent of obesity as risk factor). Individuals with SA wake up hundreds of times every night because they literally are breathless and deprived of oxygen for a few seconds. However, many people have sleep fragmentation which is unrelated to apnea. Fragmented sleep affects millions due to modern life and new technologies. SA is a process which accelerates and worsens chronic ocular disease and is considered to be a risk factor in ARMD, under the premise that hypoxia is the connection between both processes. The retina has the highest consumption of oxygen in the dark, i.e., while we sleep. During the period of apnea, the saturation of oxygen in blood can diminish over 30 points in a few seconds which, in addition to hypoxia, may cause inflammatory changes and growth factors. A recent study, carried out for tumor diseases but which can be applied to the remaining invasive neovascular processes of the body, has pointed out that sleep fragmentation, i.e., waking up many times during the night, weakens the immune system and produces chemical changes in macrophages. In the absence of continued rest, the immune system becomes weaker causing an increase in M2 macrophages and assisting vascular proliferation. A powerful immunological system and normal macrophages are essential to control neovascularization. M1 (young) macrophages exhibit different behavior than M2 (adult or aged) macrophages, and the connection between SA and ARMD is precisely the increase of this macrophage phenotype.

REFERENCES


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