Connecting the innate and adaptive immune responses in mouse choroidal neovascularization via the anaphylatoxin C5a and γδT-cells

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Neovascular age-related macular degeneration (AMD) is characterized by choroidal neovascularization (CNV). An overactive complement system is associated with AMD pathogenesis, and serum pro-inflammatory cytokines, including IL-17, are elevated in AMD patients. IL-17 is produced by complement C5a-receptor-expressing T-cells. In murine CNV, infiltrating γδT- rather than Th17-cells produce the IL-17 measurable in lesioned eyes. Here we asked whether C5a generated locally in response to CNV recruits IL-17-producing T-cells to the eye. CNV lesions were generated using laser photocoagulation and quantified by imaging; T-lymphocytes were characterized by QRT-PCR. CNV resulted in an increase in splenic IL-17-producing γδT- and Th17-cells; yet in the CNV eye, only elevated levels of γδT-cells were observed. Systemic administration of anti-C5- or anti-C5a-blocking antibodies blunted the CNV-induced production of splenic Th17- and γδT-cells, reduced CNV size and eliminated ocular γδT-cell infiltration. In ARPE-19 cell monolayers, IL-17 triggered a pro-inflammatory state; and splenocyte proliferation was elevated in response to ocular proteins. Thus, we demonstrated that CNV lesions trigger a systemic immune response, augmenting local ocular inflammation via the infiltration of IL-17-producing γδT-cells, which are presumably recruited to the eye in a C5a-dependent manner. Understanding the complexity of complement-mediated pathological mechanisms will aid in the development of an AMD treatment.

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