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Mechanisms and biomarker candidates in pterygium development

Mecanismos e candidatos a biomarcadores no desenvolvimento do pterígio

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Dear Editor:

In response to the article titled "Mechanisms and biomarker candidates in pterygium development", published in your esteemed journal, which is a well thought out and written paper, I would like to raise few points regarding this study.

Pterygium may have among its causes factors such as: changes in cholesterol metabolism, inflammation, viral infection, oncogenic proteins, lymphangiogenesis, and epithelial-mesenchymal cell transition[1]. We would like to add one more factor that may be related to the pathophysiology of pterygium.

Healthy conjunctival tissues may have Nod-like receptor pyrin3 (NLRP3). In pterygium, the NLRP3/caspase-1 pathway may be abnormally activated, accompanied by aberrant expression of IL-18 and IL-1β. There is a correlation between the number of fibroblasts and NLRP3. Activation of the NLRP3/caspase-1 pathway may be a cause of angiogenesis and fibroblast proliferation, which could induce pterygium recurrence. Nod-3 (NLRP3), caspase-1, IL-18 and IL-1β pyrodynamic receptors are common markers of pyroptosis, which is a form of programmed cell death that includes the release of inflammatory factors[2-4].

Mitomycin C use in pterygium surgery reduces the recurrence rate, suppressing angiogenesis and fibroblast proliferation, inhibiting NLRP3/caspase-1 pathway activation and the expression of inflammatory factors such as TGF-β1, VEGF, and IL-6[2-4].

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