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ABSTRACT N. PO46

INTRAVITREAL TREATMENT WITH FARICIMAB IN PATIENTS WITH AGE-RELATED MACULAR DEGENERATION.

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Background and aims

Age-related macular degeneration (AMD) is a multifactorial disorder that affects the macula leading to progressive visual loss. In developed countries AMD represents one of the primary causes of blindness among adults over the age of 60.

Faricimab is an intraocular treatment that neutralizes both the Vascular Endothelial Growth Factor A (VEGF) and Angiopoietin 2 thus stabilizing ocular vasculature ameliorating disease development and anatomical and clinical outcomes potentially for longer.

Intravitreal injection of VEGF inhibitors, has been associated with arterial thromboembolic events such as myocardial infarction and cerebrovascular accidents. Suggesting systemic adverse effects of these drugs.

Notably, numerous studies demonstrated that platelets are the main contributors to serum VEGF, which stimulates the formation of newest blood vessels. Different anti-VEGF treatments, such as bevacizumab, can be internalized by platelets, leading to an increased exposure to these treatments after platelet activation.

Cyclooxygenase 1 (COX-1) catalyses the production of a wide range of molecules, among these, thromboxane and prostaglandins. Moreover, persistent platelet activation and hyperreactivity are associated with thromboxane biosynthesis in several clinical settings.

There are conflicting reports in the literature indicating a possible correlation between COX-1 and VEGF. Most evidence demonstrated that, when COX-1 is highly expressed, we assist to a concomitant neovascularization generated by VEGF promoting pathological exudates in AMD and DME.

This study aimed at evaluating the effect of Faricimab administered in patients affected by AMD on intra-platelets levels of VEGF and COX-1.

Methods

In this study, we enrolled nine patients with AMD that underwent intravitreal treatment with Faricimab at baseline (T0) and after four months (T4).

All participants underwent clinical assessment and blood sampling. Platelets were isolated from platelet rich plasma and proteins were extracted for western blotting analysis.

Results

We observed a trend for a decrease in platelet's VEGF levels after 4 months of Faricimab treatment in all patients ($p=0.063$), except for two, where it increase. Similarly, COX-1 protein was reduced ($p=0.031$) except for the same two patients, where it increase. ΔVEGF and ΔCOX-1 (T4-T0) are directly correlated in all the patients ($rho=0.734$, $p=0.024$). Interestingly, the presence of SRPEF subretinal pigment epithelial fluid (SRPEF) was observed in these two patients, with a paradoxal increase in both platelet VEGF and COX-1.

Conclusions

Overall, the decreased tendency of VEGF within platelets at 4 months after Faricimab injection, can explain the ability of the treatment to be internalized by platelets, playing the same molecular mechanism observed for other anti-VEGF agents. Interestingly, COX-1 and VEGF protein levels in platelets directly correlate, thus, supporting a potential mechanistic link between these mediators in modulating neovascularization.

Notably, patients with SRPEF at basal – a key clinical sign for neovascular AMD – showing increased protein expression after Faricimab treatment, may represent a subgroup with suboptimal response to therapy. These findings underscore the need for larger scale studies to detect novel biomarkers of treatment response.

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