عنوان مقاله:

ANGPTL۴ suppresses the profibrogenic functions of atrial fibroblasts induced by angiotensin II by up-regulating PPARγ

محل انتشار:

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خلاصه مقاله:

Objective(s): The present study's objective was to investigate the association between angiopoietin-like ۴ (ANGPTL۴) levels and the prognosis of Atrial fibrillation (AF), the causative effect in angiotensin II- (Ang II) induced AF, and its underlying mechanisms.Materials and Methods: Baseline serum ANGPTL-۴ concentrations were measured in ۱۳۰ patients with AF. Rat atrial fibroblasts were isolated from ۱۴-day-old SD rats and transfected with Ang II treatment. Transfected cells were divided into: The control group, ANGPTL۴-OE group, Ang II group, and Ang II+ANGPTL۴-OE group. Transfected cells were used to analyze fibroblasts' proliferation, migration, and collagen production at the cellular level. RT-qPCR and western blotting evaluated the ANGPTL۴-targeted gene and PPARγ-Akt pathway.Results: In patients with AF, serum ANGPTL۴ concentrations decreased significantly compared with the healthy group. ANGPTL۴ mRNA and protein expressions were significantly down-regulated in Ang II-induced cardiac fibroblasts. ANGPTL۴ overexpression potentially attenuated Ang II-induced fibroblast proliferation, migration, and collagen production in atrial tissue. ANGPTL۴ inhibited the signaling proteins, such as PPARγ, α-SMA, and Akt.Conclusion: Our experimental data speculate that ANGPTL۴ is a key factor in regulating AF progression. Therefore, increasing ANGPTL۴ expression could be an effective strategy for AF treatment.v

کلمات کلیدی: