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Atrial Appendage Stem Cells as a Strategy to Preserve Cardiac Function Following Myocardial Infarction

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Abstract

Background: Myocardial infarction (MI) leads to extensive loss of cardiomyocytes and adverse remodeling, often progressing to heart failure. While stem cell-based approaches are continuously investigated to restore the damaged myocardium, clinical outcomes have remained modest. Cardiac atrial appendage stem cells (CASCs), which possess strong cardiomyogenic potential, represent a promising candidate for cardiac repair. Whether delivering CASCs within an elastin-like recombinamer (ELR) hydrogel scaffold enhances functional recovery following MI remains unknown.

Methods: MI was induced in eight-week-old female Sprague Dawley rats by permanent ligation of the left anterior descending coronary artery. Immediately following occlusion, MI animals were randomized to either MI+CASCs (MI+C, $2*10^6$ cells, n=9), MI+Hydrogel (MI+H, n=9), or MI+CASCs+Hydrogel (MI+C+H, n=8) administered intramyocardially in the peri-infarct zone. MI (n=8) and SHAM (n=7) operated animals served as controls. Global cardiac function and infarct severity were assessed via high-resolution ultrasound and wall motion score index (WMSI), while treatment-specific gene expression profiles were explored using clustering and heat map analysis. Data were compared by a one-way ANOVA with post hoc tests and are presented as the mean ± standard error of the mean.

Results: Following MI, systolic cardiac function was substantially declined as evidenced by LVEF (50±5% vs 81±3% in SHAM) and WMSI (1.64±0.11 vs 1.05±0.02 in SHAM). While neither MI+C nor MI+H maintained cardiac function, MI+C+H tended to preserve LVEF (64±4% vs MI) and WMSI (1.41±0.09 vs MI). MI+C+H also reduced end-systolic and end-diastolic volumes (respectively 174±36μL vs 275±38μL in MI and 452±51μL vs 541±41μL in MI), and prevented thinning of the left ventricular wall (1.8±0.1mm vs 1.4±0.2mm in MI), features not observed with monotherapies. Transcriptome analysis showed that MI+C+H downregulated genes involved in inflammation (e.g., TNF-α), fibrosis (e.g., TGF-β), and upregulated anti-oxidative pathways (e.g., GPx1 and SOD2), while monotherapies had only modest effects on these pathways.

Conclusion: Intramyocardial delivery of CASCs embedded in an ELR-hydrogel scaffold partially prevents loss of cardiac function following MI and limits structural damage better than either approach alone. These findings support the use of biomaterial-assisted stem cell delivery as a promising strategy to enhance cardiac repair after MI.