007-LIPS

"The Role of Immune Cells and Fibroblast in Mouse Myocardial Infarcted Module:

Dynamics of Inflammation and Tissue Repair"

Myocardial infarction (MI) commonly known as a heart attack, occurs when blood flow to a portion of cardiac muscle is blocked, leading to a cascade of events in the heart. This is characterized by cell death, inflammation, and ultimately fibrosis. Fibrosis can be defined as the thickening and scarring of the tissue. Immune cells such as neutrophils and macrophages are known to be pivotal in orchestrating the initial inflammatory response, while fibroblasts are critical for extracellular matrix (ECM) remodeling and scar formation. The potential interplay between immune cells and fibroblasts may be essential for the repair and remodeling following MI. This study will investigate the interaction between these cell populations and fibroblasts in mice MI tissue. Immune cell infiltration and fibroblast activation during the early (Day 3), intermediate (Day 7), and late (Day 15) phases of healing were characterized. At Day 3 post-MI, CD45 positive staining indicated significant immune cell infiltration, which may be critical for debris clearance and modulation of fibroblast activity. Concurrently, positive fibroblasts staining was observed in the infarct zone using fibroblast-specific markers. This may reflect their activation, proliferation, and migration into the damaged tissue thus initiating extracellular matrix (ECM) deposition. By Day 7, the inflammatory response had diminished, with reduced CD45-positive staining, while fibroblast staining remained prominent, highlighting their sustained role in ECM remodeling and scar formation. By Day 15, the inflammatory response had significantly subsided as the heart transitioned from the inflammatory phase to the reparative and remodeling phases of tissue healing. This research may potentially provide insight into the coordinated interactions between immune cells and fibroblasts during myocardial healing, offering potential therapeutic targets that will improve cardiac repair and prevent adverse remodeling. This knowledge can also contribute to the development of treatments that improve outcomes for patients suffering from myocardial infarction and reduce the risk of heart failure.