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Fibroblast Growth Factor 2 Affects Vascular Remodeling After Acute Myocardial Infarction

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BACKGROUND AND OBJECTIVES

• After acute myocardial infarction, vascular remodeling in the peri-infarct area is essential to provide adequate perfusion, prevent additional myocyte loss, and aid in the repair process.
• Fibroblast growth factor 2 (FGF2) is essential to the recovery of contractile function and limitation of infarct size after cardiac ischemia-reperfusion (IR) injury.
• The role of FGF2 in vascular remodeling after cardiac IR injury is currently unknown.

METHODS

Animals: Mice with a targeted deletion of the Fgf2 gene (Fgf2 KO) and wildtype controls (WT) were anesthetized with ketamine/xylazine, prepped, and ventilated by tracheostomy. Hearts were halted in diastole in potassium chloride saturated phosphate buffered saline, fixed until ST segment elevation appeared on the EKG showing LAD occlusion and was continued for 90 minutes to observe ST segment changes during ischemia and reperfusion. The suture ends were pulled apart gently to induce reperfusion, the sutures were cut close to the chest wall releasing the proximal LAD artery, and the two ends of the suture were threaded through a 0.5mm piece of PE-10 side of the chest wall and the chest wall was closed (Figure 1B). The mouse was removed from the respirator and allowed to recover for seven days. After this recovery time, mice were re-anesthetized but not ventilated.

RESULTS

• Fgf2 knockout hearts show decreased capillary density at both 3 days and 7 days after IR injury as well as increased average vessel size at 7 days of reperfusion.
• Fgf2 knockout hearts show decreased capillary density at both 3 days and 7 days after ischemia-reperfusion injury, but no significant effect on capillary size.

CONCLUSION

This study demonstrates the necessity of endogenous fibroblast growth factor 2 in vascular remodeling in the peri-infarct zone in a clinically-relevant animal model of acute myocardial infarction. These findings suggest a potential role for modulation of FGF2 signaling as a therapeutic intervention to optimize vascular remodeling in the repair process after myocardial infarction.

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