An acute myocardial infarction, the heart function of rats was restored due to the therapeutic effects of adipose derived fresh stromal vascular fraction-containing stem cells versus cultured adipose derived mesenchymal stem cells.

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Abstract

Ser/Thr kinase LRRK2 has several functional domains. LRRK2 mutations have been linked to hereditary Parkinson's disease, according to studies. Uncertainty surrounds its function in cardiovascular disease, particularly myocardial infarction. This study's objective was to investigate LRRK2's functional role in myocardial infarction. To create a myocardial infarction model, coronary arteries (left anterior descending) were cut in wild-type and LRRK2-knockout mice. Cardiomyocytes from newborn rats were exposed to hypoxia to cause hypoxic injury in culture. In mouse hearts and hypoxic cardiomyocytes, we observed elevated LRRK2 expression levels in the infarct periphery. 14 days after infarction, LRRK2-deficient mice showed lower death rates and smaller infarction areas than wild-type controls. Reduced inflammation and left ventricular fibrosis were seen in LRRK2-deficient. In the in vitro study, LRRK2 silencing decreased the activity of cleaved caspase-3, decreased cardiomyocyte apoptosis, and decreased inflammation brought on by hypoxia. However, LRRK2 overexpression boosted cleaved caspase-3 activity, raised the number of cardiomyocytes that were apoptotic, and dramatically increased inflammation brought on by hypoxia. We discovered that hypoxia increased HIF expression, which improved LRRK2 expression, when we looked at the underlying mechanisms. Through P53, LRRK2 increased the expression of HMGB1. The detrimental effects of LRRK2 overexpression following hypoxia were inhibited in cardiomyocytes when HMGB1 was blocked by an anti-HMGB1 antibody. In conclusion, LRRK2 deficiency protects the heart from damage caused by myocardial infarction. The P53-HMGB1 is involved in the mechanism causing this effect.

Keywords: Acute myocardial infarction, Adipose-derived mesenchymal stem cells, Heart function.

Introduction

Despite primary percutaneous coronary intervention, in-hospital mortality following acute myocardial infarction (AMI), particularly if complicated by cardiogenic shock, continues to be unacceptable high (PCI) This population also has high post-discharge mortality rates and readmission rates for heart failure. Therefore, the need for new, secure, and potent treatments that can enhance outcomes after AMI remains paramount importance. Numerous studies have demonstrated that cell therapy improved organ dysfunction brought on by ischemia in a variety of cardiovascular diseases. Further proof that endothelial progenitor cell (EPC)/mesenchymal stem cell (MSC) therapy effectively enhanced heart function has been provided by both experimental and clinical trials After an AMI, the treatment decreased congestive heart failure and prevented LV remodeling. Previous research revealed that adipose-derived (AD) MSCs have distinct advantages over bone marrow-derived MSCs in that they are more plentiful, less invasive to obtain, easily cultured to a sufficient number for autologous transplantation, and have higher anti-inflammatory and immunomodulatory capacity [1].

To produce sufficient numbers for cell therapy to begin, these MSCs must be cultured and expanded for at least 10 to 14 days. Therefore, these procedures may be considered impractical for use in routine clinical practise when prompt administration of ADMSCs is required to treat organ dysfunction brought on by ischemia. Therefore, it is crucial to have a therapeutic approach that is secure, has effects comparable to AMDSC therapy, and can be quickly implemented in cases of organ dysfunction brought on by ischemia The World Health Organization reported that cardiovascular diseases (CVDs) continued to be the leading cause of death in 2016, accounting

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for 31% of all fatalities. Myocardial infarction (MI) and heart failure brought on by cardiac remodeling following acute MI are significant contributing factors (AMI) Cardiomyocytes experience ischemia or infarction when the coronary artery's main branch suddenly becomes blocked. This process, known as AMI, eventually results in cardiac dysfunction and cardiomyocyte death. The prevalence of heart failure is rising due to the development of revascularization techniques, which has increased the AMI survival rate each year (HF) [2].

Numerous cardiomyocytes experience necrosis and apoptosis after an AMI, which attracts a wide array of immune cells into the infarcted heart where they consume the dead cardiomyocytes and release a variety of Proinflammatory and profibrotic cytokines. These cytokines encourage fibrotic proliferation and an inflammatory cascade reaction, which ultimately result in HF and cardiac systolic and diastolic dysfunction. The overall mortality rate due to acute myocardial infarction-induced HF remains high, despite greater awareness of these processes and the development of various interventions, and more efficient treatment methods. High mobility group box 1 (HMGB1), a common protein, can be actively secreted by stressed cells and released from necrotic cardiomyocytes when the heart suffers ischaemic injury. A typical damage-associated molecular pattern (DAMP), HMGB1 binds to a number of receptors and signalling molecules, including rage and Toll-like receptor (TLR)2/4, and causes the activation of NF-B and extracellular signal-regulated kinase (ERK)1/2 signalling, which in turn causes cells to produce proinflammatory cytokines. HMGB1 is crucial for the onset of many cardiovascular diseases, especially cardiac ischaemic injury. HMGB1 induces myocardial cell necrosis or apoptosis by increasing the expression of endothelial cell chemokine receptors and encouraging the release of inflammatory factors [3].

In experimental autoimmune myocarditis, HMGB1 blockade can reduce the degree of cardiac fibrosis. In order to treat heart damage, HMGB1 is a therapeutic target. Parkinson's disease (PD) is associated with leucine-rich repeat kinase 2 (LRRK2), a Ser/Thr kinase with multiple functional domains. Several tissue types, including the brain, heart, lung, and intestine, exhibit high levels of LRRK2 expression. A Ras complex guanosine triphosphate hydrolase domain and a C-terminal Roc domain are both present in the LRRK2 protein. In earlier research, the functional significance of LRRK2 in PD and other cerebrocortical disorders was emphasised [4].

It has recently been demonstrated that adipose-derived fresh stromal vascular fraction (SVF), which contains primitive stem cells, can be used right away with promising results in accelerating wound healing through angiogenesis and anti-inflammation (i.e., without cell culture). Based on cell surface antigens within those multipotent tissues, it was found that the adipose-derived fresh SVF contained heterogeneous populations of undifferentiated, mononucleotide elements. Thus, a simple and secure method to treat a variety of diseases has emerged using this MSC-derived uncultured/ heterogeneous SVF. The effect of SVF on LV function after AMI has, however, only rarely and with ambiguous results been discussed. In this study, we investigated the potential of adipose-derived fresh SVF therapy to restore LV function and prevent LV remodeling [5].

Conclusion

Despite the fact that myocardial bridges are typically thought to be benign, it is crucial to recognise the clinical situations in which they may cause myocardial ischemia. The most significant trigger in competitive athletes is long-lasting tachycardia brought on by arrhythmias or intense exercise. Additionally, there are some reports of myocardial ischemia brought on by coronary spasm in conjunction with a myocardial bridge in the literature. 5 For a thorough examination of symptomatic patients with myocardial bridges, multimodal imaging is crucial. Because it can reveal the location and size of myocardial bridges, CCTA is a helpful tool in the initial evaluation. Myocardial infarction caused by myocardial bridges can be distinguished from other pathologies that may coexist with tachycardia and elevated cardiac biomarkers.

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