ORIGINAL ARTICLE

Myocardial Infarction Causing Remodeling of Cardiomyoctyes

SALMAN HASEEB CHAUDHARY[1], SANA AMIN[2], TUBA JAMIL[3], SHAHID IQBAL[4], UMAIR ASGHAR[5]

ABSARCT

Aim: To observe immediate changes in cardiomyocytes repair after myocardial infarction and it's possible treatment in tertiary care hospitals of Lahore.

Methods: In this observational study 1250 patients (mean 48±5 years, 58% males) with myocardial infarction after 4 weeks. Infarcted marks in the myocardium cell enhances receptor signaling, while commencement of releasing oxygen provoke cytokine and chemokine. High population of invading leucocytes remove the infarct from dead cell area, on the other hand give boost to cells that escort to making a scar.

Results: Treatment of exact inflammatory agents through medication may not reducing the quantity of cardiomyocytes in the infracted area, but can shield the heart chamber from tissue dilatation and remodeling, which may leads to heart failure after myocardial infarction. The malfunction of medication in patients with myocardial infarction may lead to surgical intervention.

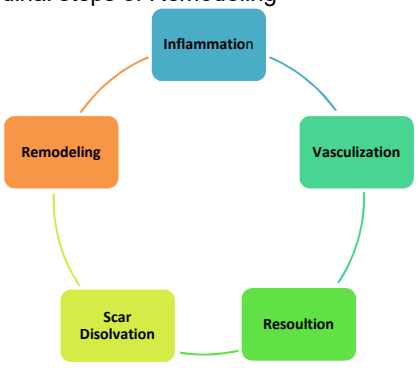
Conclusion: Treatment in response to inflammatory response to ease unconditional remodeling of cardiomyocytes in patients with myocardial infarction in quiet helpful and having good prognosis .Patients, which shows aggressive remodeling may achieve formation of new cardiac muscles.

Keywords: Myocardial infarction, Remodeling, Cardiac muscle.

INTRODUCTION

In the recent development in pharmacology and quick treatment have considerably decrease the mortality in myocardial infarction patients. Cardiac remodeling is rely on response of immune system that respond to shades of the scar on wound from dead cells and to fabricate different mediators that boost the fibroblast growth and formation of new angiogenesis[1]. Nevertheless, start of immune mediators to remodeling the cardiac muscles may not give valuable results alone[2].

Fig.1: Cardinal steps of Remodeling



[1]*PGR Medicine, SIMS, Lahore*
[2]*WMO, RHC Begowala, Distt. Sialkot*
[3]*HO, SIMS, Lahore*
[4]*Posr Graduate Resident, Cardiology, Army Cardiac Centre/CMH Lahore*
[5]*Resident FCPS Cardiology, Punjab Institute of Cardiology, Jail Road, Lahore*
Correspondence to: Dr. Salman Haseeb Chaudhry, Email: salmanhaseeb207@gmail.com

Even in growing age, the slow materialization of myocardial infarction in presence of good body health may not respond any excellent pressures directly on immune system[3].

In last 20 years a lot of research work give evidences regarding the immune reaction, which may give lengthening time to remodeling and recovery due to delay in treatment of myocardial infarction[4]. It is due quick loss of huge quantity of cardiomyocytes after myocardial infarction is due to extreme inflammatory reaction, many evidences on immune system commencement after cardiac injury is derivative from research work in ischemic heart disease[5]. However, in 1987s and 1994s widespread research evidence recommended that most of the inflammatory cells penetrate the targeted myocardium, can aggravate the ischemic injury leading towards death of extremely important cardiomyoctyes[6].

The pathogenesis in the infarcted heart, there is huge amount cardiomyocytes during sudden necrosis give many intracellular contents and initiates inflammatory process[7]. Several divergent, pathways speedup the role of inflammation after myocardial infarction[8]. In the start, harmonize flow of subcellular constituents via membrane activates[9]. Many of the experimental evidences shows continuously distruction of cardiomyocytes enhances the inflammatory response clicks the strict role of subcellular constituents in ischemic myocardium[10]. Later, damage cell area and matrix surrounding extracellular free liberate endogenous signals referred to as damage links molecular patterns

(DAMPs)[11,12]. Many hyaluronan parts, ATP, Shock protein and mitochondria may show as harmful signals in the damaged myocardium simultaneously releasing inflammatory response. DAMPs push all pro-inflammatory steps by stimulating particles of receptor family[13].

PATIENTS & METHODS

In this observational study 1250 patients (mean 48±5 years, 58% males) with myocardial infarction after 4 weeks. Infarcted marks in the myocardium cell enhances receptor signaling, while commencement of releasing oxygen provoke cytokine and chemokine. High population of invading leucocytes remove the infarct from dead cell area, on the other hand give boost to cells that escort to making a scar. As soon as infarct heals then ventricle starts to remodels, shape, cardiac cell alteration links with remodeling of post infarction cardiac cells aggravating by layer of inflammatory cells and sometimes may lead to heart failure.

All types of tissues and cell take part in remodeling of cardiac muscles and resolution of inflammatory reaction after myocardial infarction can be controlled well by advanced pharmacological agents. Because of unique cytokine and growth factor, which regulated their involvement in mononuclear cell subpopulations are suitable to act as main regulator of all the inflammatory reactions. Our study describe that T cells showing different receptors play role to boost the inflammatory response triggering remodeling of infarcted cells.

RESULTS

Many neutrophils can hold the inflammation through their death after myocardial infarction. Apoptotic neutrophils cleared by triggering huge amount of macrophages release of inflammatory mediators. Infarct area vascularize by pericyte coat of endothelial inflammatory activity.

Treatment of exact inflammatory agents through medication may not reducing the quantity of cardiomyocytes in the infracted area, but can shield the heart chamber from tissue dilatation and remodeling, which may leads to heart failure after myocardial infarction.. The malfaunction of medication in patients with myocardial infarction may lead to surgical intervention. Quick inflammation enhances the remodeling of the infracted heart through the activation of proteinases reaction , engulfing matrix loss and loose the tensile muscle force of the infarct.

All the consequence of changes explain in variables of study are mainly depends upon the aging and cardiopulmonary weakness of patients. It is exhibit by results that all the inflammatory activity after myocardial infarction have good response of remodeling cardiac cell repair.

DISCUSSION

In this observational study 1250 patients (mean 48±5 years, 58% males) with myocardial infarction after 4 weeks. Infarcted marks in the myocardium cell enhances receptor signaling, while commencement of releasing oxygen provoke cytokine and chemokine. In the start use of anti-inflammatory treatment is quiet beneficial to limit ischemic injury. Bearing in mind the important role of inflammatory response in myocardial infarction and cardiac repair, there direct role of inflammatory mediators in cardiac repair gets good results as well as new findings through exemplar shift in realistic approach[14].

In a cross-sectional study Bujak M et al, describe that targeting inflammatory mediators cannot save a mark amount of cardiomyocytes in deoxygenated infarcts, but inflammation may defend the cardiac chamber loosening and adverse dilatation, the severe irreversible condition of heart failure after myocardial infarction. After loosening sign of heart chamber dilation may affect the cardiac output for vigorous activity[15].

Carrabba N et al shows results about the pathophysiologic complications of remodeling after myocardial infarction. They describe that sudden after acute infarction, patients shows many different type of responses varies according to the age,gender, hypertension and treatment dependent on the size of infarction[16]. Few patients shows significant dilation and quick progress to heart failure as well as some patients develop progressive fibrosis leads to diastolic dysfunction[17]. They concluded that age ,gender, genetic disposition and morbid conditions (like diabetes or hypertension) might influence the remodeling of infarcted area. So these patients may focused to anti-inflammatory plans to protect from expansion of adverse remodeling[18].

In an observational study, Schwab IM et al describe the self-limited inflammation in myocardial infarction. According to them the quantitative initial assessment of different track neutophills and cytokine releases after myocardial infarction play key role in remodeling of ischemic area. The tine of inflammatory factors reaction and exudates have significant results[19]. The use of these parameters describing the resolving and the affected area during resolution phase[20]. In addition , use of different therapeutic agents develop to increase the inflammation and reducing the cardinal signs , which can decrease the the time of recovery[21].

Navarro et al gives conclusion, regarding their observational study that all the discussion regarding the acute inflammation is sensitive process in maintaining organ homeostasis[22]. So, there is no any wonder if ther is any biological process going to overcome the inflammatory process[23]. There is also process of some unique type of receptors recognized that are found on inflammatory cells that play role in sensitivity and specificity of resolving system[24]. They performed experimental process regarding inflammation and remodeling the ischemic cardiomyocytes, it has been established that resolving lipid receptors[25]. All these type of findings are giving hope regarding the opportunity for the new therapeutic and pharmacological treatment plans for good patients prognosis.

CONCLUSION

In the last the elementary role of quick inflammatory helps to maintain the poor oxygenation of ischemic cardiomyocytes and helps to provide normal homeostasis to the cardiomyocytes. Treatment in response to inflammatory response to ease unconditional remodeling of cardiomyocytes in patients with myocardial infarction in quiet helpful and having good prognosis .Patients, which shows aggressive remodeling may achieve formation of new cardiac muscles.

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