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Commentary

Clinical Trials of Refractory Angina and Cardiovascular Cell Therapy

Hong Jian*

Department of Cardiology, Zhejiang University, Zhejiang, China

DESCRIPTION

Refractory Angina (RA) is a chronic disorder that manifests as persistent angina or angina equivalents (lasting more than three months) in individuals who have objective evidence of myocardial ischemia due to untreatable Coronary Artery Disease (CAD). Such a clinical situation could get worse if Left Ventricular Dysfunction (LVD) is present. Patients with refractory angina are considered to have "no option" because they are deemed ineligible for conventional revascularization treatment due to diffuse coronary lesions, ineligibility for chronic total occlusion mechanical revascularization, or frailty due to the presence of severe comorbidities.

These pathological traits may have a significant impact on the quality of life in terms of physical function and well-being as well as midterm fatality, even if recent data have demonstrated a time-dependent rise in death rates. Risk factor reduction, strict adherence to standard medical care, and aggressive revascularization procedures have all been linked to improved outcomes.

Nonetheless, despite ongoing improvements in CAD therapy, particularly in the elderly population, the number of RA patients and the associated hospitalization expenses are continuously rising. In reality, according to the best estimates, RA affects between 600,000 and 1.8 million individuals in the USA, with an incidence of 50,000 and 30,000–50,000 new cases annually, respectively, in the USA and continental Europe. It is important to emphasize that difficult it is to provide these people with medical treatment. In actuality, there are still few treatment alternatives and available recommendations. Also, these people have unusually intricate interactions between their clinical symptoms, myocardial perfusion anomalies, coronary architecture, and ventricular function. Hence, there is an increasing demand for innovative medicines for the management of RA in individuals who are unresponsive to conventional pharmaceutical therapy and unsuitable for mechanical revascularization techniques.

Cardiovascular cell-based therapy clinical trials in RA

The field of RA has seen the execution of 26 cell-based treatment studies to far. Important elements, such as the quantity of patients treated, cell sources and kinds, baseline ejection fraction, administration techniques, and effectiveness and safety results, have been gathered and analyzed in the follows relation to the co-morbidity of Left Ventricular (LV) dysfunction, baseline EF below normal and the percentage of patients with LV impairment have been noted, when data are available.

Cell-Related parameters

Selected or unfractioned cells, mostly from Bone Marrow (BM), and to a lesser degree from Peripheral Blood (PB), and more recently from Adipose Tissue (AT), have been studied for their therapeutic potential in RA. The investigation has focused on Mononuclear Cells (MNC), endothelial progenitor cells and Mesenchymal Cells (MSC). It is important to note that there is currently no direct comparison of various cell types. Importantly, BM-derived MNC, the cell type that is administered the most frequently, have a clear safety record both in the short- and long-term. Also, information at hand revealed that BM-MNC treatment appeared helpful in reducing myocardial perfusion and angina frequency. Furthermore, the study demonstrated that repeated BM cell injections in patients who had previously responded might support therapeutic advancements. These "first generation" cell lines haven't been followed by more sophisticated "next generation" cell treatments. For instance, allogeneic treatments, which have had excellent results for other pathologic cardiac illnesses, haven't been looked into in RA. The so-called "second generation" stem cells, made up of more refined cell populations, such as Cardiac Stem Cells (CSC), cells generated from cardiospheres, or combinations of cells, have also not been studied in RA. Overall, ongoing research indicates that fresh, preliminary investigations are continuing to follow the autologous BM-MNC pathway.

Correspondence to: Hong Jian, Department of Cardiology, Zhejiang University, Zhejiang, China, E-mail: hongan@gmail.com

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