Letters to the Editor
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Improved clinical outcome after intracoronary administration of bone-marrow-derived progenitor cells in acute myocardial infarction: final 1 year results of the REPAIR-AMI trial

The final clinical analysis of the REPAIR-AMI trial is disappointing to me. The authors state that the bone-marrow-derived progenitor cells (BMC) lead to an improvement of function in the infarcted zone, especially in patients with depressed left ventricular function at baseline, increasing the ejection fraction by 5% compared with placebo. In the recent article, they concluded that BMC remained a significant predictor of favourable clinical outcome. Thus, it seems rational that the beneficial effect of stem cells may decrease the incidence of myocardial infarction, heart failure, or cardiovascular death, or the combined events. However, this was not the case. Cardiovascular death and re-hospitalization for heart failure were not different in the two groups. In the final 1 year follow-up, there was only a trend towards a lower rate of infarction in the target vessel region. However, the authors have summarized all infarctions, namely infarctions in the target vessel and in other vessels, in Table 2. More myocardial infarctions in other vessels are not likely to be related to BMC application in my view and therefore have to be listed separately. More infarctions in other myocardial regions in the placebo group correspond to more coronary dilatations in this group. Therefore, the only clinical difference results from myocardial infarctions and coronary dilatations in coronary vessels, unrelated to the targeted vessel in the trial. It is difficult to understand that stem cells injected into one coronary artery do decrease acute coronary syndromes in other coronary arteries. Analysis of combined endpoints have their own limitations and advantages. Analysis of single clinical endpoints does have more clinical relevance and may shed a clearer light into the fascinating research on stem cells.

References


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Traditional diet: hope for our children

The study by Di Salvo et al. shows abnormal myocardial deformation properties in obese, non-hypertensive children. Today, children consume a high intake of snack foods (which have N6 PUFA and transfats), confectionery (simple sugars), and carbonated drinks, and a low intake of fruits, vegetables, cereals, and milk. A high intake of N6 PUFA, transfats, MUFAs, and simple sugars all lead to an increase in triglycerides. Plasma triglycerides are a marker of oxidative stress and ectopic lipid deposition in the heart; mild intracellular diastolic dysfunction and marked deposition to systolic dysfunction. The faulty diet of children today (leading to triglyceride deposition) appears to be the most likely cause of abnormal myocardial deformation properties seen here. Limiting above faulty dietary factors and supplementing omega 3 fat would be beneficial. In addition, energy requirements can be met with short-chain saturated fatty acids (present in milk, ghee), which can be readily utilized without mitochondrial beta oxidation and hence will not be a contributor to oxidative stress that would damage the myocardium. Milk, ghee, and unhydrogenated coconut oil have been used in India safely for centuries and India had the lowest incidence of heart disease. After the switch to vegetable oils, the incidence has only been on the rise.

We need to make urgent amends in our recommendations about saturated fats as only long-chain saturated fatty acids (as well as N6 fat and transfat) lead to oxidative stress and insulin resistance. Reverting back to a traditional low fat cereal-based predominantly lactovegetarian diet supplemented by omega 3 fat and devoid of fatty, fried, refined, preserved, and processed food will pave the way for a healthy and happy future for our children.

References


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Traditional diet hope for our children: reply

We thank very much Manisha Talim for the interest in our work. We agree that the faulty diet of our children today appears to be the most likely cause of abnormal myocardial deformation properties we demonstrated in obese, non-hypertensive, children.

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