STIMULATION OF REGENERATION OF TRAUMATIC DEFECTS OF SKELETAL MUSCLE IN RATS

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Abstract: *to compare the effectiveness of various methods used to repair full-thickness muscle defects and to determine the one that gives the least amount of fibrosis and a greater number of functional muscle tissue in a given defect.*

In the damaged muscle tissue regeneration process starts normally, but because it is insufficient and slow progression, healing, includes the most part, the scarring and fibrosis. Using a variety of bioengineering structures and the fraction of stem cells, regeneration can be stimulated and the use of different antifibrotic agents, growth factors and cellular components of the process can be turned in favor of muscle recovery.

Keywords: *regeneration of muscle tissue; marcain; homologous acellular muscle design; mesenchymal stem cells; stromal vascular fraction.*

Reconstructing muscle defects following trauma is one of the problematic issues in the field of plastic surgery. Although muscle tissue is capable of regenerating, this process progresses slowly and inadequately, thus the defect is replaced with non functional fibrotic tissue [1, 2, 3].

Purpose of study is to compare the effectiveness of different methods used for reconstructing full thickness muscle defects and determine the one which yields least amount of fibrosis and greatest amount of functional tissue in a given muscle defect.

Our study has been composed of two phases. In phase 1 and 2, 20 and 25 Sprague Dowley rats were used, respectively. I-n phase 1 (group 1), full thickness muscle defects sized 16x6, 21x6, 27x6 and 32x6 mm were created on the rectus abdominis muscles of the subjects, and the muscle segments excised were re-applied as auto grafts. In phase 2 (group 2) a similar 32x6 mm full thickness muscle defect was created on the rectus abdominis muscles of each subject. In group 2a, the excised muscle was re-applied as a muscle graft before it was injected with 0.3 ml Bupivacaine (Marcaine) in group 2b, the excised muscle was treated with liquid nitrogen and boiling water. The homolog acellular muscle matrix was applied to the defect and seeded with mesenchymal stem cell suspension purified from the subject's bone marrow. In Group 2c, unseeded scaffold was used. In Group 2d, the peritoneum was left intact, other than that it was same as group 2b. In group 2e, acellular matrix was seeded with stromal vascular fraction prepared from the inguinal fat pad of the subjects. The peritoneum was left intact.

Following 4 weeks of waiting period, the muscular segments adapted to the defects were excised. Histopathological examinations were carried out under the light microscope. The areas of fibrotic and functional tissues were calculated via Image J software.

In phase 1, it was noted that group 1 d yielded the greatest amount of fibrosis, and thus its defect size was used for phase 2. When the results of phase 2 are evaluated, it is noted that the groups 2d and 2e, in which the peritoneum was left intact and a metabolic niche is provided for the cells seeding and survival yielded better functional tissue ratios. In the group in which marcaine was used, although myocytes were damaged, since satellite cells, basement membrane, vessel lumens and nerves were unaffected, the regeneration process yielded better results. When all groups were evaluated in general, despite different measures afforded, fibrosis was never altogether prevented.

In muscle damage, regeneration process starts normally, however, since it has an inadequate and slow progression, the healing concludes in greater part with scarring and fibrosis. It has been shown that using various techniques, regeneration can be stimulated and by administration of various antifibrotic agents, growth factors and cellular components, the process can be turned in favor of muscle regeneration. Further studies are needed for better understanding the complicated processes which lead to fibrosis in muscle regeneration.

References

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