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Therapeutic potential of stem cells and zinc on reduction of liver fibrosis

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Globally 1 out of 40 people died due to chronic liver diseases. In case of liver failure, transplantation is the last available therapy but due to lack of donor, graft rejection, operative damage and high cost making this therapy unsuccessful. Stem cells therapies developed new ways to treat liver diseases, but due to oxidative stress at damage site causes poor MSCs proliferation and engraftment. The aim of the current study was to explore the therapeutic potential of ZnSO₄ and MSCs on CCl₄ induce hepatic toxicity. In the current study, CCl₄ (1 µl/g) was injected intraperitoneally to female BALB/c mice, twice in a week up till four weeks to induce liver damage. MSCs was isolated from femoral and tibial bone of Balb/C mice and were cultured for two weeks. These cultured cells and ZnSO₄ both were induced separately as well as in combination in mice body. The mice were then classified into five groups: negative control, positive control, CCl₄+MSC treated group, CCl₄+ZnSO₄ treated group and CCl₄+MSCs+ZnSO₄ treated group. The morphological results showed that in contrast to only MSCs therapy, ZnSO₄ along with MSCs showed significant therapeutic result on CCl₄ injured mice. Biochemically, serum ALT and total bilirubin level were found to be significantly decreased in mice treated with ZnSO₄ and MSCs. Histopathological examination also revealed that both ZnSO₄ and MSCs have strong anti-apoptotic effect on CCl₄ injured liver by decreasing the number of apoptotic hepatocytes in both ZnSO₄ and MSCs transplanted mice. RT-PCR results at mRNA level also confirm a significant anti-fibrotic effect of ZnSO₄ and MSCs (in combination) transplanted mice on fibrotic liver as evidenced show the down-regulation of apoptotic marker (Bax) and enhancing anti-apoptotic (Bcl-xl) and hepatic marker (Albumin). Thus, it is concluded that ZnSO₄ is a powerful antioxidant and have the ability to enhance the proliferation rate of MSCs.

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