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# Trichostatin A inhibits radiation induced lung epithelial mesenchymal transition (EMT) in lung adenocarcinoma cancer cells A549

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Radiotherapy is used to treat tumors of different origins and nature. Lung cancer patients significantly dependent on radiotherapy for treatment but often lead to side effects including pneumonitis and fibrosis. It is interesting that radiation induces TGF-ß1 signaling and induces the epithelial-mesenchymal transition (EMT), is a process by which epithelial cells changes to mesenchymal cell by losing cell polarity, cell-cell adhesion and gains enhanced tumor progression capability. Our study investigated the inhibitory effect of Trichostatin A (TSA), a natural derivate isolated from genus *Streptomyces* of bacterial species, has been shown to inhibit TGF- ß1 signaling pathway, upon radiation induced lung EMT and we tried to understand the molecular mechanism using lung cancer cells A549 as a model of EMT study. The cancer cells were irradiated at 8Gy of X-ray using LINAC. The cells were divided into five treatment group untreated control (C), radiation alone (R), radiation combined with TSA (R+T) and TSA alone. Radiation induced lung EMT in A549 cells were evidenced by decreased expression of epithelial markers E-cadherin and increased expression of N-cadherin and vimentin. A marked increase in phosphor-Erk½ was observed within short span in western blot analysis. Snail protein-the master factor for EMT, which will translocate into nucleus was shown elevation after radiation treatment. Radiation group increased the migration of cancer cells whereas TSA treatment reduced the migration of cancer cells. In addition, TGF-ß1 signaling activates Smad signaling expression is elevated in radiation group and data is supported by the increased m-RNA expression of E-cadherin and snail genes. This effect was reversed by TSA treatment. In addition to this as supportive evidence we did docking which showed good interactions between snail and the TSA. Our report suggests that, TSA is effective in inhibiting TGF-ß1 pathway induced by radiotherapy.

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