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Novel Angiotensin Receptor Blocker, Azilsartan induces oxidative stress and NFkB-mediated apoptosis in Hepatocellular Carcinoma cell line HepG2

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Overexpression of renin-angiotensin system (RAS) and nuclear factor-kappaB (NF-kB) has a key role in various cancers. Blockade of RAS and NF-kB pathway has been suggested to reduce cancer cell proliferation. This study aimed to investigate the role of angiotensin II and NF-kB pathway in liver hepatocellular carcinoma cell line (HepG2) proliferation by using azilsartan (as a novel Ag II antagonist) and Bay11-7082 (as NF-kB inhibitor). HepG2 cells were treated with different concentrations of azilsartan and Bay11-7082. Cytotoxicity was determined after 24, 48, and 72 h by MTT assay. Reactive oxygen spices (ROS) generation and cytochrome c release were measured following azilsartan and Bay11-7082 treatment. Apoptosis was analyzed qualitatively by DAPI staining and quantitatively through flow cytometry methodologies and Bax and Bcl-2 mRNA and protein levels were assessed by real-time PCR and ELISA methods, respectively. The cytotoxic effects of different concentration of azilsartan and Bay11-7082 on HepG2 cells were observed as a reduction in cell viability, ROS formation, cytochrome c release, and apoptosis induction. These effects were found to correlate with a shift in Bax level and a downward trend in the expression of Bcl-2. These findings suggest that azilsartan and Bay11-7082 in combination or alone have strong potential for development as an agent for prevention against liver cancer after further studies.

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