The use of anti-angiogenesis agents in cancer treatment

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Cancer cells have an unusually high level of phosphorylation and a strong signaling effect by P53, an integrative protein in DNA repair. P53 is impaired in many forms of cancer, including liver, pancreas, colon, lung, and breast cancer.

Targeting P53 for drug development is a promising strategy to better control cancers. In 2007, P53-positive tumor cells proliferated rapidly without the misfolding of a proteasome protein.

Researchers have tested a number of compounds with P53 in the laboratory, and have identified three promising, and mostly cross-over candidates: chemokine ligands, anti-angiogenesis, and ABCK inhibitors. Early studies of P53 ligand inhibitors in multiple tumor models demonstrated clinical efficacy and favorable safety profile. A Phase I Phase I study of APC-P53 released in 2011 showed rapid growth of P53-positive colon cancer cells when dosed at 1 mg/kg.

Recently, MedQuist, an independent academic organization that finds relevant links, published a review article, The safety and effectiveness of orally administered anti-angiogenesis agents for the treatment of cancer. Anti-angiogenesis agents work by attaching DNA tags to cancer cells. These tags prevent the cancer cells from sensing the intense activity of DNA repair and repair activities between tumor cells and the surrounding tissues. The cancer cells do not detect the activity, causing increased cell proliferation.

Researchers at University of Colorado School of Medicine who were testing anti-angiogenesis agents with P53 found that anti-angiogenesis agents (Aubin cancer treatments) given intravenously to 29 patients significantly reduced the level of histologic metastasis in advanced breast cancer patients. The same treatment group was studied and treated with a drug-free placebo. Cancer cells were extracted from the tumor tissue, and purified from P53 and matched human CD4+ and CD8+ cells. Avastin and sunitinib were used, respectively. No cancer recurrence occurred, and the patients remained cancer free for 1-2 years.

Additional follow-up studies were performed with P53 antagonist-drugs during several months post-treatment. In healthy healthy volunteers, the negative reaction to anti-angiogenesis agents was found to be milder than that of chemotherapy. Patients who received anti-angiogenesis agents exhibited a significant difference in angiogenesis. The changes were expected because the same agents often caused anti-angiogenesis responses in breast cancer patients with antibodies against histological markers (inhibitors of immunohistochemistry). However, single agent P53 antagonists showed no differences in metastasis reduction. The value of P53 antagonist drugs as anticancer drugs is still to be determined. Future studies will focus on single agent P53 agonists and on possible toxicities associated with anti-angiogenesis agents.

A Fire Hydrant In The Middle Of A Forest