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Opinion Article

The Impact of Silver Nanoparticles in the Treatment of Chronic Lymphocytic Leukemia

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DESCRIPTION

Recent developments in gene expression profiling have shown that the aggressiveness and therapeutic response of Chronic Lymphocytic Leukemia (CLL) are driven by highly molecular heterogeneity. New therapeutic options have been explored in recent years with the goal of improving CLL treatment. Ibrutinib, an inhibitor of Bruton's Tyrosine Kinase (BTK), and venetoclax, an inhibitor of B-Cell Lymphoma 2 Protein (Bcl-2) are a couple of these drugs that work on several pathways that are abnormally active or upregulated in CLL. Although the majority of patients have experienced long-lasting responses to these medications, relapses can occasionally happen, particularly in the high-risk patient population, necessitating the investigation of novel therapeutic approaches. Researchers have recently become interested in nanotechnologies because of their adaptable and useful characteristics that could further enhance cancer treatment.

Because of the stability provided by the synthesis materials and the small sizes of nanoparticles, they are better able to cross biological barriers and are more easily absorbed inside of cells, which increase the cytotoxic effect of therapy. Additionally, because of their high surface-to-volume ratio, NPs can be functionalized with a particular ligand and loaded with many molecules to deliver them to cancer cells, giving them tumor-target treatment capabilities. Silver Nanoparticles (AgNPs) may be used to treat a number of neoplasms, including cervical cancer, hepatocellular carcinoma, and hematological malignancies such chronic myeloid leukemia, lymphoma, and acute myeloid leukemia, according to recent preclinical research. However, further research needs to be done to determine how they affect CLL cells and whether AgNPS may be used to create brand-new Nano therapeutic compounds that precisely target CLL cells. Nanomedicine has recently offered fresh ideas and methods that could be helpful in treating cancer. There are currently only a few studies supporting the use of Nano tools in the treatment of CLL, and nanotechnology is still in its infancy. To open up new therapy options for CLL, AgNPs with anti-leukemic activity has been developed.

AgNPs may have a better tolerability profile for a prospective application in CLL therapy given the data that they dramatically reduced CLL cell viability at concentrations lower than those employed in solid tumor cell lines. According to recent research, AgNPs in various malignancies mostly target mitochondrial integrity. In line with these findings, the AgNPs significantly shifted the Bax/Bcl-2 ratio in HG-3 cells in favor of the pro-apoptotic Bax molecule. This finding suggests that AgNPs treatment of CLL cells makes them susceptible to an intrinsic apoptosis mechanism. AgNP-induced mitochondrial-driven apoptosis in CLL cells is further supported by decreased mitochondrial membrane potential and caspase-9 activation. A well-known mechanism, mitochondrial apoptosis is sparked by a variety of biological cues, particularly intracellular Ca^{2+}. Since a dysregulated Ca^{2+} homeostasis is both a significant driver of cancer genesis and progression and a mediator affecting responsiveness to treatments, Ca^{2+} signalling plays a contentious role in CLL. In this study, the CLL primary cells displayed changed mRNA expression of several Ca^{2+} channels when compared to normal B cells, indicating that these leukemic cells are susceptible to Ca^{2+} targeting drugs has been discussed. In various tumors types, AgNPs have been found to target Ca^{2+} homeostasis. Additionally, Ca^{2+} and ROS production have a close, reciprocal interaction.

According to several studies, AgNPs have the ability to cause abnormal ROS generation, which activates the mitochondrial apoptotic pathway in a variety of cancer cell lines, including those from pancreatic, breast, gastric, and chronic myeloid leukemia. We demonstrated that, after 2 hours of treatment, when cell viability had already been significantly decreased, AgNPs caused an excessive generation of Reactive oxygen species (ROS) in HG-3 cells.

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