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Perspective

The Impact of HDAC Inhibitor and Progenitor Cells in the Treatment of Plasma Cell Leukemia

Zhen Yang*

Department of Hematology, Children's Hospital of Wenzhou Medical University, Wenzhou, China

DESCRIPTION

The characteristic of Acute Myeloid Leukemia (AML), a hematologic tumor that arises from hematopoietic stem or progenitor cells, is the clonal proliferation of immature myeloid cells in the bone marrow and peripheral circulation. At 17.6 cases per 100,000 in the US among individuals 65 years of age and beyond, AML is the most prevalent hematologic malignancy in adults. Although individuals with AML are now far more likely to survive, the disease is still incurable because of high relapse rates and drug resistance, even with the release of new medications like venetoclax.

The inability of cytotoxic therapy to eradicate Leukemia Stem Cells (LSCs) has been shown by the failure of AML treatment. Despite the possibility that LSCs could be eradicated following allogeneic stem cell transplantation using normal chemotherapy, there are two challenges: First, old and disabled patients cannot get allo-HSCT; second, a high mortality rate is caused by a number of problems such as organ toxicity, Graft-*Versus*-Host Disease (GVHD), and serious infections. These results demonstrate the therapeutic potential of LSC targeting to boost anti-AML treatment efficacy while maintaining tolerable toxicity. Ferroptosis is a type of non-apoptotic iron-dependent cell death that has garnered attention as a possible therapeutic target since it appears to be essential for cancer cell survival, particularly cancer stem cell chemo resistance. Numerous cellular metabolic processes, such as redox balance, iron management, mitochondrial activity, amino acid, lipid, and sugar metabolism, as well as other disease-related signal transduction pathways, control ferroptosis. Ferroptosis is frequently viewed as a two-edged sword in cancer cells. When cancer cells are subjected to stressful environments, a sharp rise in ferroptosis is seen as a means of maintaining homeostasis and cellular viability. However, prior research has shown that chemotherapeutic drugs in a variety of tumor's cause cell death by ferroptosis. The class of enzymes known as histone deacetylases is responsible for the removal of acetyl groups from histone proteins that bind DNA. HDACs are involved in the control of several critical physiological processes, such as cell cycle progression and apoptosis.

HDAC inhibitors are therefore attractive therapeutic medicines against cancer because they limit the aggregation of misfolded proteins and cause cell death. At present, CFDA-approved chidamide (CS055) is being used in clinical settings to treat Peripheral T Cell Lymphoma (PTCL). In AML, CS055 has also been evaluated as a monotherapy or in conjunction with treatments that target molecules. In Relapsed/Refractory Acute Myeloid Leukemia (R/R AML), clinical trials have assessed the safety and effectiveness of CS055 when combined with venetoclax and azacytidine. The class of enzymes known as Histone Deacetylases (HDACs) is responsible for the removal of acetyl groups from histone proteins that bind DNA. HDACs are involved in the control of several critical physiological processes, such as cell cycle progression and apoptosis. HDAC inhibitors are therefore attractive therapeutic medicines against cancer because they limit the aggregation of misfolded proteins and cause cell death. At present, CFDA-approved chidamide (CS055) is being used in clinical settings to treat Peripheral T Cell Lymphoma (PTCL). In AML, CS055 has also been evaluated as a monotherapy or in conjunction with treatments that target molecules.

In relapsed/refractory acute myeloid leukemia (R/R AML), clinical trials have assessed the safety and effectiveness of CS055 when combined with venetoclax and azacytidine. In conclusion, this work offers strong preclinical evidence that CS055 and chiglitazar together cause ferroptosis in leukemia stem and progenitor cells by blocking the HDAC3-SLC7A11-GSH-GPX4 pathway, thereby offering a potential therapeutic agent for the treatment of acute myeloid leukemia. This new combination regimen has a great deal of potential for clinical application in patients with refractory recurrent AML, as LSCs are the primary cause of therapy failure in AML.

Correspondence to: Zhen Yang, Department of Surgery, Children's Hospital of Wenzhou Medical University, Wenzhou, China, E-mail: yang@gmail.com

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