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Opinion Article

Assessing Renin-Angiotensin System Elements in the Circulation of Pediatric Patients with Acute Leukemia

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DESCRIPTION

Acute leukemia's are the most frequent type of malignancy in children, accounting for around 30% of all juvenile neoplasms. The pathogenesis begins with a change in the lymphocytic or myelocytic lineage cell's ability to proliferate clonally, which results in an early maturation-stage cell buildup in the bone marrow. These cells, known as blasts, build up in the bone marrow and replace the normal cell population with altered ones. About 15% of kids acute leukemias are myelocytic lineage abnormalities (Acute Myeloid Leukemia, or AML), while 80% of childhood acute leukemias are represented by lymphocytic lineage modifications (Acute Lymphoblastic Leukemia, or ALL). A minor proportion of these leukemias might exhibit biphenotypic characteristics or experience a shift in categorization while undergoing treatment. When compared to adult cases of the disease, childhood neoplasms exhibit shorter latency periods, more aggressive presentations, and faster progression.

However, they respond better to treatment and, for the most part, have a good prognosis. Anti-cancer chemotherapeutic drugs, or antineoplastics, kill cancer cells by acting systemically. Due to their high mitotic activity and short cell cycle, these chemotherapeutic drugs operate on both normal and neoplastic cells with limited selectivity, indicating their high toxicity. A little over 2% of children with ALL pass away prior to going into remission, and an additional 2% pass away during remission due to the harmful effects of the treatment. Numerous drugs that target the main molecular pathways linked to leukemia development, growth, and cell proliferation have recently been developed thanks to knowledge about the pathophysiology of the disease, genetic and molecular alterations, and mechanisms of chemotherapy resistance. One group of endogenous chemicals that has not received much research attention is Angiotensin-(1–7) (Ang-(1–7)) and Angiotensin II (Ang II), two components of the Renin Angiotensin System (RAS) that may have a direct effect on the growth of leukemic neoplastic cells. Ang-(1–7) is an endogenous heptapeptide molecule of the RAS that functions by activating the Mas receptor, one receptor, to coordinate biological responses. Exploring the function of Ang-(1–7) in hematological neoplasms is crucial because this heptapeptide can block the proliferation of many cell lines and speed up bone marrow recovery following chemotherapy-induced aplasia. Previous research has connected Ang-(1–7), via a number of mechanisms, to growth inhibition and decreased proliferation of human cancer cells as well as in xenographic tumors.

Angiogenesis, cancer-associated fibrosis, osteoclastogenesis, tumor-induced inflammation, and metastasis are all decreased by Ang-(1–7). A number of investigations demonstrated that Ang-(1–7) suppresses the proliferation of several cancer cells via lowering inflammatory prostaglandins and Cyclooxygenase 2 (COX-2) activities. By directly reducing Prostaglandin E2 (PGE-2) production in tumor tissue, COX-2 inhibition inhibits VEGF, which is responsible for promoting angiogenesis and indirectly promotes the growth and proliferation of the neoplastic cell. This result is in line with another study that showed peptides associated with the classical RAS axis increased with advancing age. Additionally, it was shown that the Ang-(1–7)/Ang II ratio and age showed a negative connection in healthy controls, supporting previously published evidence in the literature. A prior study that linked high levels of ACE activity in the bone marrow to an excessive and disordered proliferation of hematopoietic progenitors and stem cells pluripotent hematopoietic cells is consistent with the increase in Ang II concentration in leukemia patients when compared to the control group. The concept that leukemia patients have altered RAS function and homeostasis is further supported by these results.

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