Study Sheds Light on Risk of Treatment-Related Blood Cancers

Some patients develop a secondary blood cancer after receiving radiation or chemotherapy treatment for their initial cancer diagnosis

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Damon Runyon-Rachleff Innovator Elli Papaemmanuil, PhD, and colleagues at Memorial Sloan Kettering Cancer Center have uncovered new clues that may help answer a troubling question—why do some patients develop a secondary blood cancer after receiving radiation or chemotherapy treatment for their initial cancer diagnosis? Though rare, therapy-related acute myeloid leukemia (AML) or myelodysplastic syndrome (MDS) are aggressive and do not respond well to treatment. Historically, doctors thought that certain treatments caused mutations to accumulate in the blood and give rise to therapy-related cancers. The new findings suggest that the situation is more complicated but may provide a way for patients to be screened for this risk.

Tracking Genetic Changes Related to Cancer Treatment

Recently, researchers have found that 10 to 20 percent of all people over age 70 have mutations in their blood that occur spontaneously with age. A person's risk for blood cancer increases when one of these spontaneous mutations in a hematopoietic stem cell, which can develop into different types of blood cells, starts making more cells with the same genetic mutation. This process is called clonal hematopoiesis (CH).

Dr. Papaemmanuil and her team set out to define the relationship between CH in cancer patients and the risk of later developing a treatment-related blood cancer. The study included data from 24,000 people treated at Memorial Sloan Kettering and found CH in about one-third of them.

The investigators observed increased rates of CH in people who had already received treatment, especially radiation therapy and particular chemotherapies — such as platinum drugs or topoisomerase II inhibitors. Unlike the CH changes found in the general population, the CH mutations after cancer treatment occur most frequently in the genes whose protein products protect the genome from damage. One of these genes is TP53, which is frequently referred to as "the guardian of the genome."

Applying Findings to Future Treatments

To further understand their findings, the researchers conducted a three-year study including more

than 500 people, who were screened for CH before and after treatment. They found that people with pre-existing CH whose blood carried mutations related to DNA damage repair such as TP53, were more likely to have those mutations multiply after receiving cancer therapies, when compared to people who did not receive treatment.

“This finding provides a direct link between mutation type, specific therapies, and how these cells progress towards becoming a blood cancer,” Dr. Papaemmanuil said. “Our hope is that this research will help us to understand the implications of having CH, and to begin to develop models that predict who with CH is at higher risk for developing a blood cancer.”

In the future, this research may help patients by identifying individuals who are at a high risk of developing a treatment-related leukemia and finding better treatments for them. “We hope that this research will allow us to ultimately map which CH mutations a person has and use that information to tailor their primary care and also mitigate the long-term risk of developing blood cancer,” Dr. Papaemmanuil said.

The investigators also hope to use the data from this study to develop better methods for detecting CH-related blood cancers when they first begin to form — and potentially to develop new interventions that could prevent CH from ever progressing to cancer.

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