Kathy Boltz, PhD
Article below from Oncology Nurse Advisor
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Resistance to breast cancer treatment tied to mutations in estrogen receptor

Breast IMRT reduces side effects
A type of mutation that develops after breast cancer patients take antiestrogen therapies has been identified. These mutations help to explain why patients often become resistant to these types of therapies.

The discovery stems from a program at the University of Michigan (U-M) Comprehensive Cancer Center, in Ann Arbor, called Mi-ONCOSEQ in which patients with advanced cancer have their DNA and RNA sequenced to identify all types of genetic mutations that could play a role in the cancer. Researchers use the findings to help direct therapies they think will work best. But they also use the data to find new genetic links. The detailed analysis means that researchers can identify anomalies among a small number of patients.

In this case, they looked at 11 patients with metastatic breast cancer that was classified as estrogen receptor (ER)-positive, meaning the cancer is influenced by the hormone estrogen. This is the most common type of breast cancer.

The analysis found that six patients had mutations in the estrogen receptor. All of them had been treated with an aromatase inhibitor, a type of drug that blocks estrogen production. The study was published in *Nature Genetics* (2013; doi:10.1038/ng.2823).

Additionally, the researchers found that the mutations were not present before the patients started their treatment, which means it was the therapy itself that caused the mutations to develop or be selected.

"This is the tumor's way of evading hormonal therapy. These mutations activate the estrogen receptor when there is no estrogen—as is the case when a patient takes an aromatase inhibitor. It's essentially an on-switch for the estrogen receptor," said lead study author, Dan Robinson, PhD, research assistant professor of pathology at the U-M Medical School.

This on-switch essentially circumvents the effects of the aromatase inhibitor, preventing ER signaling from being shut down. That's when patients become resistant to therapy, which leaves them with few other treatment options. Some 40,000 people will die from breast cancer this year in the United States, with the majority having ER-positive tumors.

"We've been trying for a long time to understand why people become resistant to antihormone therapy. This finding sheds an entirely new light onto the problem. Now, we can look at how these estrogen receptors function and begin to develop drugs to shut down or attack this mutation," said study coauthor Anne F. Schott, MD, associate professor of internal medicine at the U-M Medical School.

The researchers also suggest that blood tests could be used to monitor patients and detect these mutations to potentially shift treatment before resistance develops. It's not yet known how frequently these mutations in the estrogen receptor occur. Currently, no treatment exists to target the mutations.

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