Biomarkers in Cutaneous Melanoma

Implications for patient education and support

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MORE THAN 100,000 NEW CASES OF MALIGNANT cutaneous melanoma (CM) will be diagnosed in the United States in 2020, with approximately 6,850 deaths projected (National Cancer Institute [NCI], 2020). About 83% of all CM is diagnosed in early stages, and localized disease has a 99% cure rate (NCI Surveillance, Epidemiology, and End Results Program, 2020). Survival rates for CM diagnosed with regional (lymph nodes) and distant metastases are not as encouraging. Despite significant treatment improvements and declining overall mortality (a 3% decrease in the death rate for all stages from 2008 to 2017), the five-year survival rate for stage IV CM is less than 30%.

However, treatment advances for metastatic melanoma, which had been stagnant for more than 50 years, have seen unparalleled progress in the last decade (Rawson & Scolyer, 2020). Understanding the biologic and genomic underpinnings of CM, as well as its interactions with the host immune system, has led to remarkable clinical advances, particularly in the identification of prognostic and predictive biomarkers and the application of targeted therapies. As such, treatment options for advanced melanoma have become quite complex and are guided by the molecular and genomic features of the individual tumor.

Oncology nurses must be familiar with the science and application of biomarkers in advanced CM that drive prognosis and treatment. A comprehensive literature review was conducted in CINAHL® and PubMed® using the following search terms: melanoma, biomarkers, targeted therapy, and immunotherapy.

FINDINGS: Targeted BRAF/MEK inhibitors, as well as immune checkpoint inhibitors, have vastly improved long-term outcomes in advanced CM. Deepening understanding of the biologic and genomic features of CM and their interactions with the host immune system is critical for predicting treatment and survival outcomes and developing new therapies.

Risk Factors

Exposure to ultraviolet (UV) radiation from sunlight plays a major etiologic role in all forms of skin cancer. UV exposure damages DNA in skin cells, and cumulative, unrepaired DNA damage can lead to carcinogenic transformation of normal cells and activation of genomic alterations, which drive malignant transformation. An estimated 46% of all oncogenic driver genomic