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Aspirin continues to surprise

Introduction

Immune checkpoint inhibitors against the PDCD1 axis have shown unprecedented clinical benefits in the treatment of refractory neoplasms. Recently, the U.S. Food and Drug Administration (FDA), approved the anti-PDCD1 (PD-1) antibody pembrolizumab for treating solid tumors with high-level microsatellite instability (MSI).

High-level MSI is commonly present in colorectal carcinomas, but not all MSI-high colorectal carcinomas respond to the immunotherapy. The oncogenesis of colon and rectal cancer represents not only a unique combination of genetic and epigenetic aberrations but also a distinct microenvironment. Thus, an important need does exist to identify predictive factors for response to immunotherapy beyond tumor MSI status, and to develop effective combination treatment strategies.

The role of aspirin as an immune checkpoint blockade inhibitor

Aspirin is a common non steroidal anti-inflammatory drug (NSAID); it inhibits PTGS1 and PTGS2. Several decades of research have provided considerable evidence demonstrating its potential for the prevention of cancer, particularly of colorectal cancer.[1]

Response to adjuvant aspirin therapy is pronounced for colorectal cancer with PTGS2 over- expression or activating PIK3CA mutations, therefore, aspirin may reduce colorectal cancer mortality by inhibiting PTGS2 and prostaglandin E2 (PGE2) synthesis that are enhanced by activated PI3K signaling, supporting the potential of these molecular alterations as tumor biomarkers.

Beyond above stated possible antitumor effects, there is evidence that refer to immune-enhancing effects of aspirin on adaptive and innate immune response.

Experimental data supporting a synergistic effect between aspirin and anti-PDCD1 antibody encouraged the accomplishment of a U.S. population-based study which suggested a stronger survival association of post diagnosis aspirin use in colorectal cancer with lower-level CD274 (PD-L1) expression than in cancer with higher-level CD274 expression. This study was the first population-based study to suggest an interaction between immune checkpoint status and PGE2 inhibition via aspirin in regulating the progression of human colorectal cancer.

These aspirin studies, unlike conventional epidemiology, elucidate differences in treatment outcomes according to patterns of molecular alterations.

Aspirin's protective effect against cancer

Daily aspirin use, has been convincingly shown to reduce the risk of colorectal cancer and recurrence of adenomatous polyps.

Blood platelets increase the levels of a protein that may support cancer cells and help them to spread. This "oncoprotein" is called c-MYC.The c-MYC regulator, controls the life-and-death cycle of cells, the synthesis of proteins, and the cells' metabolism. Researchers explain that aspirin reduces the ability of blood platelets to raise levels of the c-MYC.

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Conclusion

A systematic review demonstrated that at least 75mg of aspirin daily or every other day significantly reduced the risk of all-cause mortality in 10years and reduced the risk of CRC mortality by 33% during over a 20year period. The incidence of CRC was reduced by 40% after 10years of use.

Based on above review, The United States Preventative Services Task Force (USPSTF) has published recommendations on aspirin use for the reduction of risk of colorectal cancer (CRC).[1–8]

Moreover, aspirin is a COX-inhibitor, a group of molecules that stops the production of certain prostaglandins and mobilizes the body's immune system. Combining immunotherapies with aspirin does effectively decrease the rate of growth of bowel cancerous tumors when compared to immunotherapy alone.

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Conflict of interest

Author declares that there is no conflict of interest.

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