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Role of Inflammation in Cancer Aiding

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Perspective

The concept of an association between inflammation and cancer is more than a century old; however, the possible mechanisms linking the 2 events have recently started to be deciphered by the scientists around the world. Infection and chronic inflammation are implicated in about 25% of all human cancers worldwide and, therefore, can be prevented by controlling and targeting the inflammatory pathways that are responsible for perturbing the normal cellular homeostasis. Patients with cancer-prone chronic inflammatory and oxy-overload diseases such as Ulcerative Colitis (UC), pancreatitis, hepatitis, hemochromatosis, and Wilson disease carry a several fold higher risk of developing cancer. Inflammation in general is an important mechanism that safeguards the body against infection or injury by launching a well-coordinated immune response involving both innate and adaptive immune systems. After the elimination of the invading pathogen and wound healing, inflammation subsides. However, an unresolved inflammation owing to any failure in the precise control of immune response can continue to perturb the cellular microenvironment, thereby leading to the alterations in cancer-related genes and posttranslational modification in crucial cellular proteins involved in DNA repair, apoptosis, and cell cycle. Evidence supports the role of a number of components including cytokines, chemokines, inducible nitric oxide synthase (NOS_2), Reactive Nitrogen Species (RNS), reactive oxygen species, cyclooxygenase type-2, hypoxia-inducible factor-1 , signal transducer and activators of transcription 3, and matrix metalloproteases in inflammation-associated cancer.

An increase in the generation of reactive oxygen and nitrogen species has been implicated in the DNA alteration including point mutations in the cancer-related genes either directly or indirectly through lipid peroxidation and generation of reactive aldehyde such as 4-hydroxynonenal or malondialdehyde. A high p53 mutation load in the inflamed lesion area of the colon is correlated with an increased NOS_2 activity in the patients with UC, indicating the involvement of nitric oxide (NO●) or RNS. The risk of colorectal cancer (CRC) in patients with UC increases with the duration of the disease with a cumulative risk of 18% after 30 years. A number of genetic alterations, including microsatellite instability and mutation in TP53 tumor suppressor gene, are frequently found in UC-associated CRC. However, in contrast with sporadic CRC, mutation in TP53 occurs in early stages of cancer development in UC.

Further support and understanding for an association between inflammation and cancer in patients with UC comes from the accompanying study by Endo providing evidence supporting the role of Activation-induced Cytidine Deaminase (AID) as a link between inflammation and UC-associated CRC. AID was discovered about 9 years ago and characterized as a key molecule that is required for immunoglobulin class switch recombination and somatic hyper mutation of immunoglobulin genes to produce high affinity antibodies for specific antigens. However, AID overexpression can also have deleterious effects, including the targeting of proto-oncogenes, including BCL6 and cMYC, leading to the mutations and translocations that are associated with lymphomas. AI Dtransgenic mice are prone to lymphomas. Interestingly; AID has been associated recently with inflammation-associated cancer. The findings of the study by Endo et al5 highlight 3 major points: (1) The T-helper 2 (Th2) cytokines interleukin (IL)-4 and IL-13 can induce aberrant AID expression in human colon cells; (2) there is an increased AID expression in UC-associated CRC; and (3) there is an association between aberrant AID expression and preferential mutation in p53 tumor suppressor gene in human colonic cells [1-5].

References

1. Sarkar, Arindam, and Rajeev Kumar, Das. "Assessment of efficacy of Continuous spinal anaesthesia." J Adv Med Dent Sci Res 9 (2021): 91-94.
2. Teixeira, F, C Peixoto de Sousa, D Gonçalves and AP Pereira, et al. "70 Abdominal surgery in high-risk cardiovascular patient–anesthetic option." Reg Anesth Pain Med 3 (2021): 37-38.
3. Ferré, Fabrice, Charlotte Martin, Laetitia Boscha and Matt Kurrek, et al. "Control of spinal anesthesia-induced hypotension in adults." Local Reg Anesth 13 (2020): 39.
4. Neuman, Mark, Rui Feng, Jeffrey Carson and Lakisha Gaskins, et al. "Spinal anesthesia or general anesthesia for hip surgery in older adults." New Eng J Med 385 (2021): 2025-2035.
5. Martin-Flores, Manuel. "Epidural and spinal anesthesia." Vet Clin Small Anim Pract 49 (2019): 1095-1108.

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