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Mini Review

Chronic Inflammation Masquerading as Cancer

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Abstract

Inflammation is a body response to noxious stimuli. Cancer is a wound which do not heal. I the inflammation is progressive, persistent, chronic inflammation mediated release of chronic inflammatory mediators induced cellular and vascular changes such as cell proliferation, cell survival, angiogenesis, genomic instability, Immune modulation, invasion and metastasis leading to tumor initiation, tumor promotion, and tumor progression. That is why chronic inflammation is considered as a seventh hall mark of cancer. Where most of all cancers are preceded by chronic inflammation or infection due to external environmental factors. This article highlights about chronic inflammation and their mediators induced cellular and vascular changes leading to cancer, where chronic inflammation is mimics as cancer. In future it can be used as early cancer biomarkers, therapeutic target and agent and prognostic markers for better management of cancer.

Keywords: *NF-KB; STAT-3; Tumor Initiation; Tumor Promotion; Tumor Progression; HIF-1α*

Introduction

Inflammation is a host response to noxious stimuli such as physical or chemical or thermal [1-3]. Cancer is a major threat to mankind. Millions of patients die every year due to various types of cancer [1,2]. There is no proper advanced treatment till now for cancer with good prognosis and survival rate after many advanced treatment modalities such as surgery, radiotherapy, and chemotherapy to treat cancer [2]. Cancer is a wound which do not heal. It is a progressive, persistent, proliferation of cells after removal of stimuli. External environmental factors involved in more than 90 percent of all cancers such as tobacco, alcohol, chemical agents, chronic psychological stress, infectious agents (HPV, EBV), physical trauma. Chronic inflammation or infectious agents induced cancers are prostate cancer, esophageal cancer, head and neck cancer, oral cancer, colon cancer, pancreatic cancer, brain tumor, cervical cancer, breast cancer and ovarian cancer [2-5].

Inflammatory mediators on cancer

Acute inflammation in response to noxious stimuli involve in tissue repair and regeneration by releasing various inflammatory mediators such as IL-1, TNF-α, IL-6 and TGF-β from inflammatory cells such as neutrophils, dendritic cells, macrophages, NK cells by activating NF-kB, a key transcription factor. Some of the cytokines, which acts as anti-inflammatory cytokines are IL-2, IL-12 and IFN-δ [6-8].

If the inflammation is progressive, persistent, chronic inflammation mediated chronic inflammatory mediators such as IL-1β, TNF-α, IL-6, and TGF-βfrom mast cells, macrophages, T cells and B cells activate NF-KB and STAT-3 key transcription factors [9-11,20]. Constitutive activation of NF-Kb, a key transcription factor involve in transcription of inflammatory mediators such as cytokines, growth factors, proteolytic enzymes, involve in tumor initiation, tumor promotion, and tumor progression by cell proliferation (Cyclin d, e), cell survival (BCL-2, BCL-XL, survivin), genomic instability (ROS, RNS, AID, arginase1), immune modulation (IL-4, IL-5, IL-10, iL-13, IL-17), angiogenesis 9VEGF, Cox-2, IL-8, HIF-1α), invasion and metastasis (Mmp's2, 9, UPA) [12-14]. NF-Kb, a key transcription factor acts as an antagonist to p53, a tumor suppressor gene, a guardian of genome mutated in more than 50 percent of all cancers by ROS, RNS, NO. Chronic inflammation is considered as a seventh hall mark of cancer involved in cell proliferation, cell survival, tissue damage, genomic instability, immune modulation and invasion and metastasis [15-17].

Conclusion and Future Perspective

Chronic inflammatory mediators involved in all stages of tumor initiation, promotion, and tumor progression can acts as an early cancer biomarker, therapeutic and prognostic markers for better management of cancer [18,19].

Anti-inflammatory agents such as vitamin A, B, C and anti-inflammatory cytokines such as IL-2, IL-12 and IFN-δ, also anti-inflammatory drugs involve in preventing and suppressing the inflammatory process for further progression and future therapeutic purpose for better management of cancer. There is no treatment till now, which can kill only cancer cells without killing normal cells. 25 percent of all cancers are associated with chronic inflammation or infection. Chronic inflammation is considered as seventh hall mark of cancer.

Bibliography

1. Freddie B., *et al*. "Global cancer statistics 2018: Globocon estimates of incidence and mortality worldwide for 36 cancers in 185 countries". *CA: A Cancer Journal for Clinicians* 68.6 (2018): 394-424.

2. Shrihari TG. "Dual role of inflammatory mediators in cancer". *Ecancermedicalscience* 11 (2017): 721.

3. Coussens LM and Werb Z. "Inflammation and cancer". *Nature* 420.6917 (2002): 860-867.

4. Grivennikov S I., *et al*. "Immunity, inflammation and cancer". *Cell* 140.6 (2010): 883-1013.

5. Glanben L., *et al*. "Chronic inflammation and cytokines in the tumor microenvironment". *Journal of Immunology Research* (2014): 149185.

6. Nathan C. "Points of control in inflammation". *Nature* 420.6917 (2002): 846-852.

7. Fernandes JV., *et al*. "The role of the mediators of inflammation in cancer development". *Pathology and Oncology Research* 21.3 (2015): 527-534.

8. Philip M., *et al*. "Inflammation as a tumor promoter in cancer induction". *Seminars in Cancer Biology* 14.6 (2004): 433-439.

9. Blackwill F and Mantovani A. "Inflammation and cancer: Back to Virchow?" *Lancet* 357.9255 (2001): 539-545.

10. Candido J. "Cancer-related inflammation". *Journal of Clinical Immunology* 33.1 (2013): 579-584.

11. Ioannis LA., *et al*. "How do cytokines trigger genomic instability?" *Journal of Biomedicine and Biotechnology* (2012): 536761.

12. Brett B., *et al*. "Tumor induced perturbations of cytokines and immune cell networks". *Biochimica et Biophysica Acta* 1845.2 (2014): 182-201.

13. Korniluk A., *et al*. "From inflammation to cancer". *Irish Journal of Medical Science* 186.1 (2016): 57-62.

14. Mantovani A and Sica A. "Macrophages, innate immunity and cancer: balance, tolerance and diversity". *Current Opinion in Immunology* 22.2 (2010): 231-237.

15. Shrihari TG. "Inflammation related cancer-Highlights". *Journal of Carcinogenesis and Mutagenesis* 7 (2016): 1-2.

16. Lippitz BE. "Cytokine patterns in patients with cancer: A systematic review". *Lancet Oncology* 14.6 (2013): 218-228.

17. Shrihari TG and Ramesh DNSV. "Chronic inflammation induced immunosuppression in Tumor microenvironment of oral cancer". *Global Journal of Medical Research* 16 (2016): 1-8.

18. Facciabene A. "Tumor hypoxia promotes tolerance and angiogenesis via CCL28 and Treg cells". *Nature* 475.7355 (2011): 226-230.

19. Masako N and Daniel WS. "Multifaceted roles of PGE2 in inflammation and cancer". *Seminars in Immunopathology* 35.2 (2013): 123-137.

20. Oian BZ and Pollard JW. "Macrophage diversity enhances tumor progression and metastasis". *Cell* 141.1 (2010): 39-51.

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