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Mini Review

Can we Detect Cancer Early? How Early?

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Abstract

Cancer is a major threat to mankind, mainly caused by external environmental factors such as tobacco, alcohol and chemicals. Cancer cells work like normal cells. Normal cells to become cancer cell, it must undergo tumor initiation, tumor promotion, tumor progression. Cancer cells circulate through out human body by seeding, in this case can we detect the cancer early. How early? This article highlights about controversies related to early detection of cancer.

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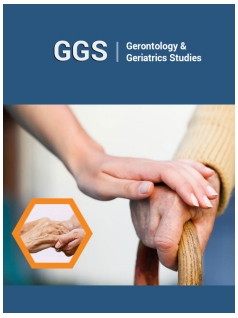

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Introduction

Cancer is a major threat to mankind, mainly caused by external environmental factors such as tobacco, alcohol and chemicals. Cancer cells and normal cells work alike, we cannot kill cancer cells without killing normal cells (Albert zen gyorgi). We daily exposed to external environmental factors by food we consume, adverse habits, air we inhale, exposed to many chemicals. If we scan for cancer cells in our human body, we have many cancer cells but not all cancer cells will turn into clinical cancer. Normally cancer cells are destroyed by our immune system but in cancer, cancer cells are escaped from immune system by its specialized mechanism called as immune evasion [1-4]. When do we detect cancer early when the cancer cells circulate throughout the body and seeding every corner of human body? If the normal cells to become clinical cancer the cells have to undergo three stages

1. Tumor initiation
2. Tumor promotion
3. Tumor progression

In tumor initiation stage the normal cell to become cancer cell it has to undergo mutation, not one mutation is enough, but 4-5 mutations are required to turn into cancer cell. In tumor promotion the cancer cells undergo cellular proliferation and cell survival and become dormant for many years until further activating factors provoke the process. In tumor progression the progression of cancer by angiogenesis, invasion and metastasis by various inflammatory mediators such as cytokines(IL-1,TNF-α,IL-8,IL-10,TGF-β,IL-6), chemokine's(CXCL,CXCR,CCL types), growth factors(EGF,FGF,VEGF) and proteolytic enzymes(up A, MMP's) secreted from inflammatory cells such as neutrophils, macrophages, and mast cells activate a key transcription factors NF-KB and STAT3 involved in tumor progression [2-9]. Most of all cancers, more than 90 percent of all cancers are due to external environmental factors such as chewing or smoking form of tobacco, alcohol, chemicals ingestion (such as lead, arsenic, silica), infectious agents (HPV, EBV), nutritional deficiency, and chronic psychological stress.

External environmental factors induced inflammatory cytokines, chemokines, growth factors; proteolytic enzymes from inflammatory cells activate transcription factors, which in turn activates inflammatory mediators involved in tumor progression. Hence, chronic inflammation is considered as a seventh hall mark of cancer. 25 percent of all cancers are due to chronic inflammation or chronic infection [1-5,10-13]. Chemokine's are chemotactic cytokines recruits immune cells to the site of inflammation. Chemokines are involved in tumor progression by expression of its receptors on leucocytes produced by tumor and stromal cells. Chemokines involved in recruitment of neutrophils are CXCL1, CXCL2, CXCL3, CXCL5, CXCL6, CXCL7, CXCL8, chemokines involved in recruitment of dendritic cells, macrophages, natural killer cells by CCL2, CXCL12-CXCR4, CCL4, CCL5, MCP-1. Chemokines involved in recruitment of lymphocytes and natural killer cells by CXCL12-CXCR4, CXCL9, CXCL10, CXCL11, CCR7-CCL21, CXCL19, CCL21 [1-17].

Inflammatory mediators produced from neutrophils, macrophages, mast cells such as IL-1, TNF-α, and COX-2, microbial agents activate NF-KB a key transcription factor, IL-6, EGF, FGF, PDGF, IL-10 activate STAT-3 transcription factor. NF-KB a key transcription factor induced expression of inflammatory mediators involved in cell proliferation by activation of cyclin D, cyclin E cell cycle regulatory proteins, cell survival by BCL-2 and BCL-XL anti-apoptotic protein, angiogenesis by IL-8,COX-2,VEGF, immune modulation by IL-4,IL-5,IL-13,IL-10,TGF-β, genomic instability by ROS,RNS free radicals, ions, AID (Activation induced cytidine deaminase) enzyme, invasion and metastasis by UPA (Urokinase plasminogen activator), MMP's 2,9 (Matrix metal proteinases 2,9) all these changes leads to tumor progression.

STAT-3 transcription factor involved in cell proliferation by activation of cyclin D, cyclin E cell cycle regulatory proteins, cell survival by activation of BCL-2, BCL-XL anti-apoptotic proteins. Both NF-KB and STAT-3 key transcription factors work together involved in tumor progression [18,19]. HIF-1α (Hypoxia induced factor-1 Alfa) transcription factor produced by tumor associated macrophages in hypoxic tumor microenvironment acts as a transcription factor for IL-8, COX-2, VEGF involved in angiogenesis and immune modulation. If all these factors taken into consideration are, we are doing more harm than good in the process of detecting the cancer early.

References

1. Grivennikov SI, Greten FR, Michael K (2010) Immunity, inflammation and cancer. Cell 140(6): 883-899.
2. Shabnam S, Michael K (2015) Immunity, inflammation, and cancer: an eternal fight between good and evil. J Clin Invest 125(9): 3347-3355.
3. Shrihari TG (2017) Dual role of inflammatory mediators in cancer. Ecancermedicalscience 11: 721.
4. Korniluk A, Koper O, Kemona H, Piekarska VD (2016) From inflammation to cancer. Ir J med sci 186(1): 57-62.
5. Chai EZ, Siveen KS, Shanmugam MK, Arfuso F, Sethi G (2015) Analysis of the intricate relationship between chronic inflammation and cancer. Biochem J 468(1): 1-15.
6. Shrihari TG (2018) Inflammation and cancer: Critical view. Journal of analytical oncology 7(2): 32-36.
7. Shrihari TG (2018) Inflammation related cancer or cancer related inflammation. The European research journal 4(1): 1-5.
8. Shrihari TG (2018) Cytokines major molecular messengers orchestrate between inflammation and cancer. The European research Journal 4(3): 140-144.
9. Shrihari TG (2017) Endorphins on cancer: A novel therapeutic approach. J Carcinogen Mutagen 8: 298.
10. Shrihari TG (2017) Inflammation-related cancer or cancer-related inflammation. The European Research Journal 4(1): 1-5.
11. Sethi G, Shanmugam MK, Ramachandran L, Kumar AP, Tegaonkar V (2012) Multifaceted link between cancer and inflammation. Biosci Rep 32(1): 1-15.
12. Shrihari TG (2016) Inflammation related cancer- highlights. J Carcinog Mutagen 7: 269.
13. Shabnam S, Karin M (2015) Immunity, inflammation, and cancer: an eternal fight between good and evil. J Clin Invest 125(9): 3347-3355.
14. Chai E Z, Siveen KS, Shanmugam MK, Arfuso F, Sethi G (2015) Analysis of the intricate relationship between chronic inflammation and cancer. Biochem J 468(1): 1-15.
15. Sethi G, Shanmugam MK, Ramachandran L, Kumar AP, Tegaonkar V (2012) Multifaceted link between cancer and inflammation. Biosci Rep 32(1): 1-15.
16. Shrihari TG (2018) Inflammation and cancer-critical view. Journal of analytical Oncology 7(2): 2-4.
17. Okada F (2014) Inflammation related carcinogenesis: current findings epidemiological trends, causes and mechanisms. Yonago Acta Med 57(2): 65-72.
18. Diakos CI, Charles CA, Mcmillan DC, Clarke SJ (2014) Cancer related inflammation and treatment effectives. The lancet oncology 15(11): 493-503.
19. Glauben L, Fuente PTM, Chanitra T, Marcela AH (2014) Chronic inflammation and cytokines in the tumor microenvironment. Journal of immunology research 3: 1-19.