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Short communication

Inflammation: Cancer's Friend or Foe?

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ABSTRACT

Inflammation is a response to noxious stimuli. Acute inflammation induced release of inflammatory mediators involved in protective role by repair and regeneration. Chronic progressive, persistent inflammation induced release of chronic inflammatory mediators involved in tumor progression by tissue damage, DNA damage, Gene mutation, cell proliferation, cell survival, invasion and metastasis by activation of a key transcription factor such as NF-KB. This article briefs about the acute and chronic inflammatory mediator's protective and promotive role in tumor.

Keywords: NF-KB; Cytokines; Growth factors; Acute inflammation; Chronic inflammation

INTRODUCTION

Cancer is a complex disease of mankind due to external environmental factors such as tobacco, alcohol, chemical agents and viruses. These external agents induce inflammatory mediators such as IL-1, TNF-α, COX-2, and TGF-β, EGF from acute inflammatory cells such as neutrophils, macrophages, and mast cells involve in regeneration and repair. Low dose of RNS and ROS free radicals acts as antibacterial activity produced by neutrophils and macrophages. CD8 T cells involved in antitumor activity by producing IFN-γ. Dendritic cells are antigen presenting cells presents antigen to T cells, which activates B cells to produce antibodies involved in anti-tumor activity.

NK cells are innate immune cells first innate immune cells to activate during viral infection and cancer produce IFN-γ, opsonin, and granzyme-B involved in antiviral and anti-tumor activity. TGF-β in early stages of cancer produced by macrophages involved in antitumor activity.IL-2 and IL-12 cytokines are produced by macrophages and CD4 T cells involved in antitumor and anti-inflammatory activity. If the inflammation is chronic progressive, persistent results in dysregulated inflammatory mediators such as cytokines, chemokines, growth factors, enzymes are released from inflammatory cells such as macrophages, B cells and mast cells results in chronic inflammation, tumor initiation, tumor promotion, and tumor progression [1-7]. IL-2, IL-12, IFN-γ cytokines produced by immune cells such as macrophages, neutrophils are anti-tumor cytokines. Early stage of cancer TGF-β acts as anti-tumor activity whereas in later stages it acts as protumor activity. IL-4, IL-5, IL-10, IL-13, IL-17 acts as protumor cytokines in chronic inflammatory tumor environment induced tissue damage with immune modulation properties.IL-1,TNF-α,IL-6,EGF activates NF-KB whereas FGF,IL-11,IL-6 activates STAT-3 transcription factor. Tregulatory cells (Tregs) formed from Th1 cells mediated by TGF-β release IL-10 involved in immune modulation. Innate and adaptive immunity is suppressed by immune modulating factors such as IL-10, TGF-β, Tregs [8-10].

HIF-1α transcription factor for IL-8, COX-2, and VEGF. Protumor cytokines acts as tumor prognostic markers and antitumor cytokines acts as antitumor therapeutic agents will be usefull for tumor immunotherapy. CD4 T cells involved in antitumor activity involved in cell mediated immunity. CD8 T cells are cytotoxic T lymphocytes involved in antitumor activity. B cells involved in humoral immunity activate plasma cells to release antibodies acts as antiviral and antitumor activity.

Whereas Bregs (regulatory B cells) are mediated by TGF-β release IL-10 involved in immune modulating properties [11-14]. Chronic inflammatory mediators such as IL-1, TNF-α, IL-6, and EGF activate NF-KB a key transcription factor. NF-KB, a ubiquitous transcription factor present in all cells in cytosol as an inactive form activated by inflammatory mediators such as IL-1, TNF-α, EGF, LPS (Lipopolysaccharide) [8-13]. Activation of NF-KB transcription factor in immune cells involved in immune

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cell development, activation and maturation [14-17]. Dysregulated NF-KB transcription factor in cells involved in tumor progression by activation of inflammatory mediators results in cell proliferation by cyclin D, E, cell survival by BCL-2,BCL-XL, angiogenesis by IL-8,COX-2,VEGF,HIF-1α, genomic instability by ROS,RNS,AID(Activated cytidine deaminase), Arginase 1, immune modulation by TGF-β,IL-4,IL-5,IL-13,IL-10,Tregs (Regulatory T cells), invasion and metastasis by UPA(urokinase plasminogen activator), MMp's 2,9 (Matrix metallo proteases) [17-20]. Chronic inflammation is considered as a seventh hall mark of cancer, which accounts 25 percent of all cancers.

CONCLUSION

Dual action of inflammation includes in acute inflammation, the inflammatory mediators are anti-inflammatory and anti-tumorigenic, in chronic inflammation, the dysregulated chronic inflammatory mediators involved in tumor progression by activation of NF-KB and STAT-3 transcription factors. Chronic progressive, persistent inflammation induced release of chronic inflammatory mediators involved in tumor progression by tissue damage, DNA damage, Gene mutation, cell proliferation, cell survival, invasion and metastasis by activation of a key transcription factor such as NF-KB.

CONFLICT OF INTEREST

None

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