Inflammation Related Cancer - Highlights

Shrihari TG*
Department of Oral Medicine and Radiology, Krishna Devaraya College of Dental Sciences and Hospital, Bangalore, India
*Corresponding author: Shrihari TG, Department of Oral Medicine and Radiology, Krishna Devaraya College of Dental Sciences and Hospital, Bangalore, India, Tel: 91-9844386188; E-mail: drshrihariomr@gmail.com
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Abstract

Inflammation is a body response to infectious or physiological agents. During this process aggrevated chronic inflammation mediated release of various mediators such as chemokine's, cytokines and growth factors by neutrophils, lymphocytes, and macrophages are predominant abundant cells in tumor microenvironment promotes tumor progression. Myeloid derived suppressor cells are immature myeloid progenitor cells upon activation of chronic inflammatory mediators release various factors such as COX2, INOS, ROS, Arginase1 bring about immunosuppression and tumor progression. This article highlight about inflammation related cancer mediated by macrophages and myeloid derived suppressor cells.

Keywords: Inflammation; Cancer; Myeloid derived suppressor cells; Inflammatory mediators

Abbreviations


Introduction

Inflammation is a physiological response to injury or infection. In this process various chemical mediators are released by recruited inflammatory cells such as cytokines, growth factors, reactive oxygen species, nitrogen species and inflammatory signals are activated as a defensive action to combat against infection and heal injured tissue [1-3]. Inflammatory mediators are released by inflammatory cells such as neutrophils, macrophages, lymphocytes. If the inflammation is persisted and aggravates chronically, results dysregulated immune system in tumor microenviroment results in initiation, promotion, progression of cancer [2,4].

Inflammatory-immune cells, stromal tissue affect malignant cells through production of chemokin's, cytokines, growth factors, prostaglandins and reactive oxygen and nitrogen species [2,3].

Infection and chronic inflammation contributes 10-20% of cancer. Preclinical and Clinical studies suggested that dysregulated immune system in Chronic inflammation is epidemiologically related to cancer [3,5,6]. Some inflammatory conditions or injury that are associated with malignancy are Lichen planus, gingivitis and chronic periodontitis associated oral squamous cell carcinoma, Sialadenitis related salivary gland carcinoma, Gastric acid associated Barrett's metaplasia and reflux oesophagitis associated oesophageal carcinoma, Sjogren's syndrome and Hashimoto's thyroiditis associated mucosa associated lymphoid tissue lymphoma, UV radiation associated skin inflammation melanoma, Silica, asbestos, smoking associated silicosis and bronchiitis associated lung carcinoma [7]. Neutrophils pro-tumoral activity by releasing ROS(Reactive oxygen species) and RNS(Reactive nitrogen species) induce genetic instability, promote angiogenesis by VEGF(Vascular endothelial growth factor), MMP-9 or prokinetin2 (BV8) and neoplastic cell invasion by mediators such as HGF(Hepatic growth factor) and oncostatin [8,9]. Macrophages are the predominant immune cells in tumor microenvironment contributes to tumor progression by releasing chemokin's, cytokines, growth and transcriptional factors such as VEGF, PDGF, EGF, FGF, TNF-Alfa, IFN -Beta promotes tumor growth [8]. IL-10, TGF-Beta, CCL17, CCL18, CCL22, PGE2, ROS, IDO, B7-H1 antibody promotes immunosuppression by naïve T cell energy, Recruitment of Th2 cell, inhibition of Th1 cell and induction of Treg (T regulatory) cell. VEGFs, PDGF, EGF, FGF, TGF-Beta, MMP-9, CXC18, ELR+ chemokine, cyclooxygenase promote angiogenesis and lymphangiogenesis. MMPs, TGF-Beta, UPA, UPAR, VEGFs, PDGF, Cathespins, TNF-Alfa, MCP-1, M-CSF, SR-A, HIF-1 promotes matrix remodeling, Cell proliferation, resistance to apoptosis, epithelial mesenchyme transition, tumor
invasion and metastasis by activating master transcriptional factor NF-
KB. T lymphocytes produce cytokines such as IL-2, IL-4, IL-6, IL-10,
TNF-Alfa, IFN-Gamma, COX-1 and COX2 promote malignant
disease. IL-17 cytokine generated by CD4+Th17 cells produce
inflammatory mediators such as TNF-Alfa, IL-6 and IL-1 Beta
promoting cytokines, promote tumorigenesis by production of
PGE2, VEGF, keratinocyte-derived chemokine and macrophage
inflammatory protein-2 (MIP-2) angiogenic factor [10-12].

Myeloid derived suppressor cells (MDSC) are heterogeneous
population of immature myeloid cells that are precursors of dendritic
cells, macrophages and/or granulocytes derived from bone marrow is
of two types granulocytic or monocytic. Myeloid derived suppressor
cells has a potent regulatory immune response and have a major role in
chronic inflammation and tumor development by activation of tumor
derived mediators or cytokines such as IL-1beta, IL-4, IL-6, IL-10 and
TGF-Beta induce expression of arginase-1, Inducible nitric oxide
synthase (iNOS) or ROS immunosuppressive factors which can initiate
apoptosis in T cells, Programmed cell death and immunosuppression
of effector cells such as adaptive and Innate immune cells [13-15].

Conclusion

Expansion of Myeloid derived suppressor cells by factors such as
GM-CSF, G-CSF, M-CSF, Stem cell factor and VEGF. Myeloid derived
suppressor cells activates STAT3 and MMP (Matrix metalloproteases)
there by promoting angiogenesis and invasion, cell Proliferation by
further activation of STAT3 induces the secretion of bFGF and VEGF.
Myeloid derived suppressor cells activates matrix metalloproteases
facilitate cancer cell invasion and intravasation by disruption of
endothelial cadherins, degradation of extracellular matrix, adhesion
proteins or basement membrane vessels.

MDSC also facilitate epithelial to mesenchymal transition in cancer
cells by using factors such as epidermal growth factor (EGF),
Hepatocyte growth factor (HGF) and TGF-Beta [16,17].

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