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Opinion

Defensive or Offensive Role of Inflammation in Cancer

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Inflammation is a defensive mechanism against noxious stimuli such as physical or chemical or thermal injury. During inflammation various mediators are released such as chemokines, cytokines, enzymes and growth factors from inflammatory innate and adaptive immune cells for regeneration and repair. Chemokines are chemotactic cytokines released from inflammatory cells such as leucocytes involved in recruitment of immune cells to the site of inflammation. Cytokines are inflammatory mediators secreted by innate and adaptive immune cells such as neutrophils, macrophages, mast cells, dendritic cells, NK cells, T cells, and B cells. Growth factors such as EGF, FGF, VEGF, produced by immune cells involved in cell proliferation, cell survival, angiogenesis, by activating transcription factors NF-KB and STAt-3 work together.

Proteolytic enzymes such as Mmp's2, 9 (Matrix metallo proteases 2, 9), UPA (urokinase plasminogen activator) involved in extracellular matrix degradation induced tissue damage. IL-8, TGF-β, COX-2, acts as dual role in inflammation induced cancer. During acute inflammation, the first immune cells recruited to the site are neutrophils later followed by macrophages, mast cells, Dc's, Nk cells by chemokines release inflammatory mediators such as IL-1, TNF-α, IL-6, PDGF involve in cell regeneration by cell proliferation, antibacterial action of ROS, RNS free radicals at low concentration, collagen deposition by activation of NF-KB, a key transcription factor. IL-2, IL-12, and IFN-ϒ are anti-inflammatory, antiviral, and antitumor activity.

PAMPs (pathogen associated molecular patterns) are specialized recognition by pattern recognition receptors (PRR) belongs to TLR (Toll like receptors) and DAMPs (Damage associated molecular patterns) activate Nf-kb, a key transcription factor.

Constitutive dysregulated, chronic progressive, persistent inflammation can cause cellular and vascular changes by activation of Nf-KB, a key transcription factor in chronic inflammation results in various cellular and vascular changes by transcription of inflammatory mediators involved in cell proliferation, cell survival, angiogenesis, immune modulation, genomic instability, and invasion and metastasis. HIF-1α transcription factor for IL-8, COX-2, and VEGF involved in angiogenesis in hypoxic tumor microenvironment. High levels of ROS, RNs free radicals released from chronic inflammatory cells such as macrophages, mast cells and inflammatory cytokines such as IL-8, TNf-1α involved in gene mutation, cell injury, tissue damage, cell aging, and cell death. IL-4, IL-5, IL-13, IL-17 pro-inflammatory cytokines produced by chronic inflammatory cells such as macrophages, T cells, involved in immune modulation and tissue damage.

Tregs (T regulatory cells) mediated TGF-β induced immune modulation by releasing IL-10. Immune modulatory action of Bregs (B regulatory cells) by releasing IL-10. NF-Kb, a transcription factor acts opposing the action of P53, a guardian of genome mutated by inflammatory mediators such as RNS, ROS, arginase 1 and AID (Activated cytidine deaminase).

Understanding of acute and chronic inflammatory microenvironment and their mediators, interaction with other immune and epithelial cells, actions helps in preventive, therapeutic and prognostic purpose in management of cancer patients.

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