Clinical Surgery Journal

Short Communication | Vol 4 Iss S6

Systems of Failure of Immune Response to Early Carcinogenesis Recharacterize such Early Carcinogenesis

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Received: January 18, 2021; Accepted: January 31, 2020; Published Date: February 10, 2020

ABSTRACT

The characterization of injury to normal cells in terms of an early stage in carcinogenesis is a re-characterization of the modulated stage of emergence of a failed immune response to such early carcinogenesis. It is significant to consider modulatory steps in the evolution of the neoplastic lesion in terms that beget further suppression of the immune system to survey the landscape modulatory potentials for further growth of the tumor and its often quasi concurrent mechanisms of spread within the body. It is therefore in terms of a significant concurrent series of events that formulation of the early carcinogenesis is inherently coupled with the events of a suppression of the immune response as re-characterized systems of essential emergence of progressiveness.

KEYWORDS

Tripartite motif (TRIM); Apoptosis; Innate immunity; Autophagy; Inflammation; Autoimmunity; Tumor development

INTRODUCTION

The conceptual approach to treat established tumors rather than to prevent tumorigenesis is a fundamental consideration within the domains of progressive evolution of given tumors that expand and grow towards the metastatic spread of the lesions. Tripartite motif (TRIM) family proteins, most of which have E3 ubiquitin ligase activities influence intracellular signaling, development, apoptosis, protein quality control, innate immunity, autophagy and carcinogenesis [1]. Such concept is further important as enveloping deterministic considerations of the presence of tumor antigens that are also often expressed by normal tissues.

Alarmins are involved as initiators and participants in host defense, gene expression regulation, homeostasis, inflammation, autoimmunity and oncogenesis [2]. In such terms, the emergence of tumor lesions is a realization of a malignant transformation of initially normal tissues, but normal in relative consideration only. cGAS-STING has dichotomous roles in tumor development and immunity [3]. The evolution of normal tissues is an evolving concept in its own right, within the further relative relevance as a source for tumors that spread in different terms. The incremental dimensions of realization of tumor growth and spread are themselves only relatively related in that

Citation: Lawrence M Agius, Systems of Failure of Immune Response to Early Carcinogenesis Recharacterize such Early Carcinogenesis. Clin Surg J 4(S8): 21-24.

inceptive neoplasms may spread at initial stages of evolution of these lesions.

INCREMENTAL EVOLUTION

The incremental, evolutionary development of tumors is a deterrent in the development of cancer vaccines that are to a significant extent a measure aimed at eradicating the tumor at a specific developmental stage of growth and spread of the lesion. Inflammation predisposes to the development of cancer and promotes all stages of tumorigenesis [4]. In such terms, the further participation of injury to actively dividing and also dynamically stationary tumor cells is a concept based only on theoretical considerations. Intestinal microbiota are linked to colorectal carcinogenesis in terms of inflammation induction, genotoxin biosynthesis interfering with cell cycle regulation, and the production of toxic metabolites [5]. The human microbiome is an emerging target in cancer development and therapeutics and is directly oncogenic through mucosal inflammation or systemic dysregulation [6]. The relative dimensions of the evolutionary character of a given tumor lesion are therefore of important import within the dynamic landscape of a mitotic lesion that continually and forcefully evades the immune system reactivities. It is significant to consider the vaccination program proposed for eradication of a given tumor lesion as a simplistic concept with regard to the emergence of neo-antigens that further propel the carcinogenesis program of growth and spread of the lesion.

STAGES OF EMERGENCE

It is further to such considerations to realize that the emergence of tumors constitutes in itself a fundamental step in the evolutionary program of the immune response to act against the initial steps of carcinogenesis. Occasionally, immune-mediated elimination of human Papilloma virus infection of cervical cells fails with increased expression of the E6 and E7 oncoprotein resulting in disrupted cell cycle, cell proliferation and malignant transformation, and induced immunosuppression [7]. It is significant to realize that the immune response is at early stages of tumorigenesis a failed immune response to the cancer lesional evolution. Virally infected cells can acquire cancer hallmarks, especially after induced immunosuppression and exposure to co-carcinogenic stimuli [8]. The importance of such considerations is the direct projection of carcinogenesis as terms of reference to such early failure of the immune responses to eradicate such given tumor lesion.

The realization of an injurious insult to early transforming events in carcinogenesis is the failure to suppress the malignant transformation events at the early inceptive stage of lesion development. In such terms, emergence of carcinogenesis as a progressive evolution is tantamount realization of events that persist indefinitely within systems of modulation of the immune system.

IMMUNE RESPONSE

The immune response is itself a biological determinant at early stages of carcinogenesis within simple evolutionary terms of systems of biologic modification based largely on dynamic interactivities with the emerging tumor lesion. Immune evasion by tumors involves targeting the regulatory T cell function or secretions, antigen presentation, modifying the production of suppressive immune mediators, tolerance and immune deviation [9].

The emergence of carcinogenesis is a consideration in terms of the failed constitutive response of the immune system in its attempts to eradicate the neoplastic lesion. Tumor immunosuppressive networks include regulatory T cells, natural killer T cells and distinct subgroups of immature and mature dendritic cells [10]. The pervasive evolutionary course of the tumor is thus a characteristic of many essential facets of the failure program of an otherwise potentially responding immune system in early stages of carcinogenesis.

DEVELOPMENTAL WAVES

Developmental waves of progression of the tumor lesion are additional factors in the projection of metastatic spread in terms of evolution of the neoplasm. The full realization of a carcinogenesis is persistent during the whole evolutionary course of the lesion.

The carcinogenesis phenomenon is a fully operative basis for the progressive evolutionary course of a neoplastic lesion that gains potential for further progressive growth and spread within the body. It is significant to consider evolution as a dynamic and reactive adaptation to accommodating facets of the immune response. CD4 (+) CD25 (high) T regulatory cells (Treg) are important mediators of active immune evasion in cancer [11]. The emergence of such significant import further translates the immune response in terms of modulatory influences that are themselves characterizations of the intrinsic early carcinogenesis phenomenon. Some recent studies have demonstrated that myeloid-derived suppressor cell targeting improves the efficacy of immune checkpoint blockade in cancer therapy [12].

MODULATION

Such considerations are suggestive of the presence of an all pervasive and dominant phenomenon that modulates both the early carcinogenesis process and the modulated failure of the immune response such as early stages of the carcinogenetic process. In such terms, the biologic basis of the immune response is a modulated and re-characterized the process of mutual suppression of both T and B lymphocytes.

Such a concept is suggestive of a suppression and dominant phenomenon within the genetic landscape dynamics of the given tumor lesion. Perforce reinterpretation of dynamics of tumor evolution is significant in terms of the essential process of emergence of the tumor lesion as the concurrent failure of an immune response as re-characterized essential features of the early carcinogenesis process. This is a term of reference within simple project models of the evolutionary course of a given neoplasm as encompassed derivation of the lesion from originally normal tissues of origin of the tumor.

Tumor-related suppression mechanisms include accumulations of adenosine-reducing regulatory T-cells, release of suppressive micro-vesicles by tumor cells, and expression of toll-like receptors on the tumor cell surface [13].

Such a conceptual background for the early carcinogenesis may elucidate the nature of tumorigenesis as both progressive and persistently capable of spread within a body system of nonresponse of the immune reactivities. It is significant to recognize such a phenomenon that is inherently persistent in terms of the failures of the immune reactivities and responses in suppressing the tumorigenesis growth and spread of the given lesion.

CONCLUDING REMARKS

The uniformity of failure dynamics of the immune response as a permissive failure to suppress the tumorigenesis is a strict characterization of the lesion as fundamentally mutual evolutionary steps in carcinogenesis. The emergence step in such carcinogenesis is a strict re-characterization of immune biology in terms of the modulation of the malignant transformation step. The emergence, in turn, of conceptual redefinition of such malignant transformation is the fundamental character of progressiveness of a lesion that is potent modulator and re-modulator within the evasive dimensions of the tumor lesion processes of growth and spread within immune system non surveillance dynamics. The perforce contributions of such failed immune surveillance are permissive in terms arising from the dynamics of recognition and reactivity of the immune system. In terms, therefore, of overall consequence, the emergence of early stages of carcinogenesis incorporate

the nature of failure of the immune response to suppress tumorigenesis as emergent process of malignant transformation of essentially normal tissues in first instance.

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