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Inflammation in Renal Cancers: The Invisible Killer

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Introduction

Several studies have been conducted to investigate the role of inflammation in tumorigenesis and cancer progression. To establish an inflammatory tumour microenvironment, neoplastic cells, as well as surrounding stromal and inflammatory cells, engage in well-coordinated reciprocal interactions. The tumor-associated inflammatory tissue is highly plastic, capable of changing its phenotypic and functional characteristics on a continuous basis. Chronic inflammation appears to play a critical role in the development of urological cancers, according to mounting evidence. The mechanisms that drive tumour initiation, growth, progression, and metastasis are discussed in this review of the origins of inflammation in urothelial, prostatic, renal, testicular, and penile cancers. We also talk about how tumor-associated inflammatory tissue could be used as a diagnostic marker for clinically significant tumour progression risk and as a target for future anti-cancer therapies.

Although inflammation is a self-limiting host defence strategy against biological, chemical, and physical agents, it is also thought to be a hallmark of cancer development. Inflammation, especially persistent inflammation, stimulates cell proliferation and local host response, resulting in cell damage and the development of various diseases, including cancer. Furthermore, the tumour microenvironment, which is rich in cytokines, chemokines, transcription factors, and immune cells, can promote tumour growth and immune escape. Inflammation plays an important role in the tumour story, and oncogenesis is thought to be associated with chronic infection and inflammation in 15-20% of cancers. Helicobacter pylori, hepatitis B or C, and autoimmune diseases have all been linked to gastric and colorectal cancer, hepatocellular carcinoma, and mucosa-associated lymphoid tissue lymphoma.

Numerous studies in recent years have found that inflammatory molecules and pathways promote the development of various cancer types, including genitourinary tumours. Chronic inflammation is recognised as a risk factor in bladder cancer (BC), alongside other well-established causes such as smoking and occupational exposure to aromatic amines. Although advanced age, African descent, family history, and genetic mutations are known risk factors for prostate cancer, the mechanisms underlying its initiation and progression remain unknown. As potential causes of PCa, inflammation-induced cellular stress and repeated genomic damage have been investigated. Evidence is accumulating that immune cells and inflammatory pathways can promote renal cell carcinoma growth and immune escape [1].

Description

Whereas there is insufficient evidence to suggest that inflammation plays a role in other urogenital tract tumours, such as testicular and penile cancer. By examining the various signalling pathways and downstream transcription factors, we examine how inflammatory molecules cause cell transformation, tumour cell proliferation, and metastasis in genitourinary tumours. We also discuss the ability of tumor-associated inflammatory cells to cause tumour immune evasion and future prospects for the use of novel anti-inflammatory agents and the mechanisms underlying their anticancer activity.

Although the inflammatory response appears to be important in the occurrence and development of tumours, its role in carcinogenesis remains unclear. Pathogens, diet, mechanical and chemical trauma, and other factors can all trigger an inflammatory response. To describe how inflammation is related to cancer, two models have been proposed: an intrinsic pathway induced by DNA damage, chromosomal instability, and epigenetic changes, and an extrinsic pathway associated with inflammatory signals caused by autoimmune diseases or infections. Both of these pathways are characterised by the activation of transcription factors that drive the inflammatory cascade, such as Nuclear Factor-B and Signal Transducer and Activator of Transcription (STAT)-3. Overall, the most common causes of genitourinary inflammation are infectious and noninfectious in nature [2].

Schistosomiasis is a known risk factor for BC, which is the most common cancer type in men and the second most common cancer type in women in Sudan, Egypt, Sub-Saharan Africa, and Yemen. Squamous cell carcinomas account for 60-90% of all schistosomiasis-associated BC, with adenocarcinomas accounting for 5-15% and urothelial carcinomas accounting for the remainder. Schistosoma haematobium eggs cause an inflammatory response, which frequently produces genotoxic factors that cause genomic instability and tissue damage. Intravesical instillation of Schistosoma haematobium antigens causes inflammation and urothelial dysplasia in CD-1 mice, according to in vivo studies. It has been proposed that Schistosoma haematobium causes cancer by inducing K-RAS mutations [3,4].

The link between bacterial infections and the development of BC is still debatable. Some authors have found that patients with a history of recurrent urinary tract infections are more likely to develop BC, whereas fewer UTI events treated with antibiotics are associated with a lower risk of BC. In contrast, Jiang et al. discovered a significantly lower risk of BC in patients with recurrent UTIs, which could be explained by the antimicrobial treatment's anti-cancer effect, higher exposure to nonsteroidal anti-inflammatory drugs, and the immune response induced by bladder infection. Gram-negative uropathogens are the most common pathogens in bacterial prostatitis, but Gram-positive and atypical microorganisms can also be responsible [5].

Conclusion

Inflammation is closely associated with cancer and plays an important role in tumour development and progression. A microenvironment rich in inflammatory cells, growth factors, and DNA-damaging agents causes tissue injury and mutation accumulation in epithelial cells, promoting their growth. In turn, the mutated cells produce cytokines and recruit inflammatory cells, forming an inflammatory tumour microenvironment that aids in angiogenesis, migration, and metastasis. Because the expression of inflammatory mediators is higher in tumours than in normal tissues, using anti-inflammatory drugs alone or in combination with chemotherapy could provide a valuable contribution to cancer prevention and treatment.

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Conflict of Interest

There are no conflicts of interest by author.

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