

Human Cytomegalovirus and Colorectal Adenocarcinoma: Any Association?

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Abstract

Background: Human cytomegalovirus (HCMV) has been implicated to transform many mammalian cells, so we tried to investigate whether HCMV participates in human colorectal carcinogenesis.

Methods: Immunohistochemistry analysis using monoclonal antibody of HCMV early protein was conducted on tissues of 32 colorectal adenocarcinomas and eight colorectal hyperplastic polyps, tissues of normal tumor margin were considered as control.

Results: HCMV early protein was detected in five out of 32 (15.6%) colorectal adenocarcinomas, while none of the eight

colorectal hyperplastic polyps and tissues of normal tumor margins was positive for the virus early protein.

Conclusion: the data of this study suggests that HCMV may participate in the process of colorectal carcinogenesis as it is evidenced that HCMV was detected in colorectal cancer tissues only.

Key wards: HCMV, CRC.

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Introduction

Human cytomegalovirus (HCMV) is a member of the herpesvirus family that is a ubiquitous human pathogen worldwide. In the United States, 40% to 90% of adults are seropositive owing to exposure to the virus at some time during life^(1, 2). CMV infection is lifelong, and the latent virus can reactivate to cause serious illnesses when the host is immunocompromised^(3, 4). Various *in vitro* studies have demonstrated that the gene products of CMV are capable of modulating cell cycle progression and apoptosis by regulating the expression of a number of important host genes⁽⁴⁻⁷⁾. For example, CMV infection has been shown to transcriptionally activate the expression of the proto-oncogenes *c-fos*, *c-jun*, and *c-myc*^(8, 9).

The immediate early viral proteins also can block the induction of apoptosis by tumor necrosis factor α or the adenovirus E1A proteins⁽¹⁰⁾. In addition, CMV has been shown to transform a variety of mammalian cells that are tumorigenic in nude mice⁽⁵⁾.

Detection of an infectious agent in human cancers might have important implications in cancer treatment and prevention. To further study whether CMV participates in human colorectal carcinogenesis, we examined colorectal hyperplastic polyps, adenocarcinomas, and normal-appearing colonic mucosa for the presence of one of the HCMV early proteins.

Material and Methods

The specimens included in the study were paraffin embedded blocks of 32 colorectal adenocarcinomas with their normal tumor margin tissues and 8 colorectal hyperplastic polyps. 5 μ m tissue sections on positive charged slides were subjected for immunohistochemistry analysis to detect HCMV early protein using

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