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β-Catenin as a Biomarker in Diagnosis of Tumors with Special Emphasis on Colorectal Carcinoma

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Introduction

Historically, clinicopathology (histopathology and cytopathology) is a test that used in diagnosis of tumors. This technique has been evolved from Haematoxylin and Eosin (H&E) based staining to Immunocytochemistry (IHC) to evaluate tumor histogenesis and subtype [1]. Most tumor biomarkers are protein produced by both normal and cancerous cells. However, they are made at much higher rate in cancerous cells. Some biomarkers are specific to one type of cancer, whereas others are associated with more than two cancer sites. No unique tumor marker can detect a specific site of cancer [2]. Recently, genomic profile of individuals with tumors has shown mutations in up to 100 protein encoded genes [3]. Tumor biomarkers played big role in diagnosis of cancer screening, during follow up therapy and after treatments [4]. The challenge of future importance of biomarkers will be not only facilitating diagnostics and therapeutics promises but also strengthen the guidance for implementation of cancer prevention strategies as well as the development of personalized medicine [5].

Objective

The current article aimed to investigate the impact of β-catenin expression as well as immunohistochemical and clinicopathological features in diagnosis of colorectal carcinoma patients. The relation among these issues in cancer progression may in turn be helpful in development of a novel strategy to control colorectal carcinoma.

β-catenin

β-catenin is one of many useful tumor biomarkers identified of internal malignancy. It is a protein encoded by the CTNNB1 gene. Its function is regulating the coordination of cell–cell adhesion (sticking cells together), gene transcription and in communication among cells [6]. Over expression of β-catenin were found in various cancer sites (colorectal, lung, malignant breast tumor and hepatocellular carcinoma). Furthermore, β-catenin is also involved in cell signaling as an important part of Wnt signaling pathway. Wnt signaling pathway initiates the division of cells, which is involved in cells development before birth. β-catenin contents of several repeats, each approximately 40 amino acids long, all these repeats twisted together into a single, rigid protein called Armadillo (ARM) domain [7-11].

Colorectal Cancer (CRC)

A malignant tumor (neoplasm) arising from the inner wall of the large intestine (colon): According to the World Health Organization (WHO), cancer killed nearly 8 million people each year. The four most common cancers occurring worldwide are lung, female breast, colorectal and prostate cancer. Colorectal cancer is the third most common malignancy in developed countries. Moreover, it is the second most frequent cause of cancer-related death [12,13]. Researchers have found several risk factors that might increase a person's chance of developing colorectal polyps or colorectal cancer. Some colorectal cancer risk factors can be control (diet, overweight, physical inactivity, smoking and heavy alcohol use), other cannot be controlled (age and family history). Mutation in β-catenin gene in colorectal cancer cells resulted in changes of protein expression. Results demonstrated that gross alterations in the cellular distribution of β-catenin in primary colorectal cancers, as well as in the metastatic tumors. These changes may be the consequence β-catenin gene mutations [14].

Furthermore, results showed that β-catenin moved from the cytoplasm to the nucleus, where it may serve as a transcriptional factor to stimulate tumor development (uncontrolled cells division). Such finding suggested that nuclear translocation of β-catenin is involved in development of intramucosal cancer rather than adenoma [15,16]. Nevertheless, it was reported that genetic mutations that initiated and drove the progression of colorectal cancer caused defects in signalling pathways that consequently affected cell function. The Wnt signalling pathway is a key pathway that is disrupted in colorectal cancer and β-catenin was the key mediator of Wnt signalling. Deregulation of Wnt/β-catenin signaling was a hallmark of the majority of sporadic forms of colorectal cancer and resulted in increased stability of the protein β-catenin. β-catenin was then shuttled into the nucleus where it activates the transcription of its target genes [17,18].

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