Invasion and Migration in Colorectal Cancer Patients

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Brief Report

Colorectal cancer (CRC) is the third most prevalent cancer in the world, with rates of occurrence and mortality continuously increasing in Asia, Europe, and North and South America. CRC is the second largest cause of death worldwide, according to GLOBOCAN, with an estimated 9.6 million deaths each year. According to accumulating data, stage 1 CRC has a 5-year survival rate of over 90%; whereas, stage 4 CRC has a 5-year survival rate of about 10%. Furthermore, around 25% of all CRC patients still experience symptoms as a result of the illness or its spread.

Genetic changes in the colonic epithelium lead to the transformation of normal epithelium into an adenoma, then in situ carcinoma, and finally invasive and metastatic tumours. Because CRC is a genetically diverse disease with a wide range of mutations, patients are likely to benefit from targeted therapy for specific molecular abnormalities. Importantly, it has become clear that advances in molecular staging provide a source of prognostic and predictive information to the traditional staging method, which divides CRC patients into four prognosis categories based on the original tumor's size. The existence or lack of metastases, as well as the involvement of regional lymph nodes. Since a result, identifying clinical indicators responsible for monitoring CRC treatment is critical, as this might lead to the creation of novel therapeutic targets, lowering the risk of mortality in CRC patients.

CEACAM1 (carcinoembryonic antigen-related cell adhesion molecule 1) is a cell adhesion molecule that belongs to the carcinoembryonic antigen family and is a subgroup of the immunoglobulin (Ig) superfamily. It was formerly known as CDA or biliary glycoprotein. CEACAM1 is a tumour suppressor that also serves as an apoptosis regulator. CEACAM1 expression is reduced in cancerous tissues originating from the colon, breast, prostate, and endometrial. In individuals with cutaneous malignant melanoma and lung adenocarcinoma, however, overexpression of CEACAM1 is linked to a shorter survival time and metastatic dissemination. Colorectal adenomas and adenocarcinomas had lower levels of CEACAM1 mRNA than colorectal adenomas and adenocarcinomas. Recent studies have found that CEACAM1 protein expression is upregulated in adenocarcinomas, and that the intensity of the whole CEACAM1 protein is linked with TNM stage, but not with overall survival or disease-free survival. The cause for CEACAM1 re-expression in colorectal cancer has yet to be established.

LncRNAs, a new kind of functional RNA, are made up of nucleotides and are mostly produced by RNA polymerase II (Pol II). Many lncRNAs have been linked to 30 human illnesses, including malignancies such breast cancer, liver cancer, glioblastoma, and leukaemia. ANCR is a lncRNA that is located on chromosome 4 of the human, upstream of the *USP46* gene, and contains MIR44 and SNORNA between its first and second introns. ANCR is claimed to govern a variety of biological processes, including cell proliferation, transcriptional regulation, differentiation, invasion, and metastasis, using several mechanisms such as signals, molecular decoys, scaffolds for protein-protein interactions, and enhanced RNAs. Enhancer of zeste homolog 2 (EZH2) is a histone methyl transferase with carcinogenic characteristics; overexpression of EZH2 promotes neoplastic transformation by enhancing cancer cell proliferation and invasion. By regulating EZH2 and runt-related transcription factor2, ANCR has been demonstrated to control osteoblast development and increase bladder cancer spread in previous research (Runx 2). To the best of our knowledge, no research has been done on the role of lnc RNA-ANCR and EZH2 in CRC progression regulation. As a result, we postulated that ANCR expression was linked to EZH2, and we set out to explore the expressions of ANCR and EZH2 in CRC, as well as the impact of lncRNA ANCR on CRC cell invasion and migration via modulating EZH2 expression.

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