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Is Endocan a Novel Prognostic Marker for Colorectal Cancer?

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Abstract

The present study aimed to investigate the relationship between pretreatment serum Endocan levels and pretreatment serum VEGF levels in colorectal cancer cases, as well as colorectal cancer prognosis.

Keywords: Colorectal cancer; Endocan; VEGF

Introduction:

Colorectal cancer is the second most common type of cancer in women and the third one in men. In terms of worldwide prevalence, it ranks third [1]. The prognosis of colorectal cancer is dependent on the stages in the TNM system. The development of tumors and metastases depend on a delicate balance between endogenous angiogenic factors, which cause the formation of new blood vessels, and anti-angiogenic factors [2]. The process of angiogenesis consists of a multitude of sequential and interconnected steps including positive and negative regulators [3]. Today, it is known that angiogenesis is not only essential for tumor growth but also is responsible for the cancerous transformation of a premalignant tumor, circulation of cancer cells, and the transformation of micro-metastases into typical metastatic lesions [4]. Without doubt, the vascular endothelial growth factor (VEGF) is the most important molecule that plays a role in the angiogenetic process [5,6]. VEGF does not only induce the proliferation of endothelial cells but also increases the vascular permeability and causes the formation of a fibrin matrix that enables stromal cell invasion by increasing the extravasation of proteins through tumor vessels [7]. The data provided by preclinical and clinical studies indicate that VEGF is the predominant angiogenic factor in colorectal cancer [8]. A positive correlation was detected between increased VEGF levels and lymph node involvement, and distant organ metastasis [9].

Endocan (Endothelial cell-specific molecule-1) is a 50-kDa dermatan sulfate released from activated vascular endothelial cells including tumor cells [10]. In humans, it is mostly secreted by the lung endothelium, followed by renal and gastrointestinal channel endothelia with lower rates [11]. Endocan plays a role in the regulation of a series of biological processes such as adhesion, migration, proliferation and neovascularization [12]. Recent studies suggest that endocan expression is associated with tumor neovascularization, angiogenic transition in stem cells, and endothelial-mesenchymal transition process such as arterial wall remodeling [13]. Endocan synthesis is increased by VEGF-A, VEGF-C, IL-1, TNF-a, TGF-B1, and FGF-2; and it is reduced by interferon-γ and phosphatidylinositide 3-kinases (PI3K) [10].

The present study aimed to investigate the pretreatment Endocan levels in colorectal cancer cases with their VEGF levels, as well as its relationship with prognosis.

Methods:

This study included a control group of 16 individuals and a patient group of 67 colorectal cancer cases who referred to the Medical Oncology Outpatient Clinic at Izmir Katip Celebi University Ataturk Research and Training Hospital from January 2012 to December 2015. The patient's pre-treatment Endocan and VEGF levels, as well as body mass index (BMI), body fat index, fat-free body mass, and total body water scores were recorded.

We formed a control group of healthy individuals with similar demographic characteristics and measured their body mass index (BMI) scores, Endocan and VEGF levels [14]. The inclusion criteria were as follows: No pregnancy or breastfeeding, no other malignancies except colorectal cancer and no record of prior treatment for colorectal cancer.

We received their verbal informed consent prior to the study. The BMI was calculated in kg/m². Anthropometric measurements and bioelectric impedance analysis of the patients were performed at the beginning (basal). Body composition [total body water (TBW), fat- free mass (FFM), fat mass (FM), percent body fat] was measured by bioelectrical impedance analysis using TANITA BC-420MA scale. One nurse performed the measurements for all patients.

Results:

Twenty-six of the patients had rectal cancer, and 41 of them were diagnosed with colon cancer. Regarding the stage at diagnosis, 1 patient was Stage-1, 12 were Stage-II, and 25 were Stage-III. The patient group's mean age was 60.6, which was 52.8 for the control group. The mean weight of the former was 69.2 kg and BMI was 25.4, which were 76.1 kg and 26.1 for the latter.

In the follow-up period, 43 patients presented metastasis at the onset or during the follow-up, while 24 patients presented no metastasis or progression (tumor-free patient group). Endocan and VEGF levels of metastatic patients were 10.43 ± 2.59pg/mL and 304.2 ± 314.07pg/ ml respectively. No significant difference was find between the patient and control groups in terms of height, weight, age or BMI levels. The examinations showed no significant difference between the groups except the VEGF level. A comparison of two groups with respect to VEGF levels revealed a significant difference (p: 0.040). No significant difference was observed between the groups in terms of Endocan levels. Table 2 presents the serum VEGF, Endocan levels, body composition and anthropometric measurements of patients and the control group.

Conclusion:

In this study we found pre-treatment serum VEGF levels in themetastatic patient group significantly higher than both the tumor- free patient group and the control group. A comparison of tumor-free colorectal cancer cases with the control group showed no significant difference in terms of VEGF levels. These findings support the idea that high VEGF levels could be associated with poor prognosis. An assessment for a cut-off value to indicate poor prognosis revealed no threshold VEGF level to anticipate prognosis. In the present study, although a negative correlation was observed between VEGF levels and overall survival, the difference was not significant.

References:

1. Ferlay J, Soerjomataram II, Dikshit R, Eser S, Mathers C, et al. (2014) Cancer incidence and mortality worldwide: sources, methods and major patterns in GLOBOCAN 2012. Int J Cancer 136: E359-E386.

2. Jemal A, Center MM, DeSantis C, Ward EM (2010) Global patterns of cancer incidence and mortality rates and trends. Cancer Epidemiology Biomarkers Prev 19: 1893-1907.

3. Folkman J (1990) Endothelial cells and angiogenic growth factors in cancer growth and metastasis. Cancer Metastasis Rev 9: 171-174.

4. Daly ME, Makris Reed M and Lewis CE (2003) Hemostatic regulators of tumor angiogenesis: a source of antiangiogenic agent-