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Effect of IGF-1 on Bladder Cancer

Mehmet Aydin Dagdeviren
Gaziantep University

Mehmet Ozaslan
Gaziantep University

Abstract: Insulin-like Growth Factor 1 (IGF-1) is a protein involved in cell growth, development, and regeneration, and it is produced in the liver in response to growth hormone. Recent studies have shown that IGF-1 may play a role in the development and spread of certain types of cancer. While IGF-1 promotes cell proliferation, it can also inhibit apoptosis a feature that may lead to uncontrolled growth of cancer cells and aggressive tumor progression. Numerous studies have investigated IGF-1 levels in relation to breast, prostate, colorectal, and lung cancers. Researchers have found strong evidence that IGF-1 not only contributes to cell proliferation in cancer patients but may also facilitate metastasis. Therefore, research targeting IGF-1 levels and its receptors is considered a promising strategy in cancer treatment and management. In our study, we examined IGF-1 levels in 100 bladder cancer tissue samples 50 from patients with low-grade cancer and 50 from those with high-grade cancer. Tissue samples were properly processed and converted into supernatant form, after which they were analyzed using the chemiluminescent immunoassay method on an autoanalyzer. According to our results, the IGF-1 levels in patient samples were found to be below the reference range of the IGF-1 kit used. However, statistical analysis revealed that IGF-1 levels were significantly higher in patients with high-grade bladder cancer compared to those with low-grade cancer. This suggests that IGF-1 expression pathways may be disrupted in cancer and could have a negative impact in high-grade cases. We believe that our study may shed light on cancer by determining the level of IGF-1 hormone in tissue samples of patients with bladder cancer.

Keywords: IGF-I, CPS, Apoptoz, Chemilimmunoassay, Supernatant

Introduction

Insulin-like Growth Factor 1 (IGF-1) is a protein that plays a crucial role in cell growth, development, and regeneration in the body. IGF-1 is produced in the liver in response to growth hormone (GH) stimulation and regulates various biological processes. However, in recent years, increased research into the effects of IGF-1 on cancer cells and its relationship to cancer development suggests that IGF-1 could be a new target in cancer therapy (Pollak, 2000; Giovannucci et al., 2003). While IGF-1 stimulates cell proliferation and growth, this process can accelerate and become uncontrolled in cancer cells. IGF-1 promotes cell division and can speed up the growth of cancer cells (Le et al., 1998).

Normally, cells are expected to undergo a controlled cell death mechanism known as apoptosis in response to genetic damage or abnormal conditions. However, IGF-1 can inhibit apoptosis, preventing cell death. This feature may allow cancer cells to survive and proliferate, leading to more aggressive tumor growth (Baserga, 2003). Numerous studies have revealed that elevated levels of IGF-1 are more common in certain types of cancer. The effects of IGF-1 vary depending on the type of cancer. For example, high levels of IGF-1 have been associated with an increased risk of common cancers such as breast cancer, prostate cancer, colorectal cancer,

and lung cancer. IGF-1 particularly stimulates the growth of estrogen receptor-positive breast cancer cells (Yu & Rohan, 2000).

Furthermore, studies on prostate cancer show that high IGF-1 levels can increase the risk of prostate cancer and contribute to more aggressive tumor growth (Giovannucci et al., 2003). IGF-1 is also reported to play a role in the development of colorectal cancer, with high IGF-1 levels accelerating tumor growth and spread in colorectal cancer patients. While IGF-1 promotes the growth of lung cancer cells, its role in lung cancer can vary depending on the tumor stage and type. IGF-1 can also facilitate the metastasis of cancer cells. It promotes cell migration, which can accelerate cancer spread. Studies showing that metastatic cancer cells are influenced by IGF-1 levels suggest that IGF-1 should be targeted in cancer treatment. The effects of IGF-1 on cancer are crucial in developing treatment strategies. Treatment methods that target IGF-1 receptors aim to inhibit cancer cell growth and stop tumor spread. Drugs that inhibit IGF-1 receptors can slow down or stop tumor growth in certain cancer types, offering a new opportunity for cancer therapy (Baserga et al., 2003).

Additionally, controlling IGF-1 levels may help reduce cancer risk. Lifestyle factors such as obesity, age, diet, and exercise play an important role in regulating IGF-1 levels. Conditions like obesity can increase IGF-1 levels and raise cancer risk (Giovannucci, E 2003). In conclusion, while IGF-1 is a critical factor for cell growth and development, it is also an important molecule associated with cancer development. IGF-1's ability to promote cancer cell growth, inhibit apoptosis, and facilitate metastasis demonstrates the need to target IGF-1 in cancer treatment. However, the effect of IGF-1 on cancer is complex and can vary across different cancer types. Controlling IGF-1 levels may help reduce cancer risk, and future research will allow us to better understand IGF-1's role in cancer and improve cancer treatment by targeting IGF-1.

Method

In the pathology laboratory, paraffin tissue samples from 50 low-grade and 50 high-grade bladder cancer patients (total of 100 patients) diagnosed with bladder cancer were obtained from the laboratory archive. After the tissue underwent the paraffinization process, supernatant was obtained using Atl tissue homogenizer solution. The IgF supernatants were analyzed using the chemiluminescent immunoassay method on an Immulite 2000 device

Results and Discussion

The study was conducted. In the control group, the measurement at low levels was 773,692 CPS, while the measurement at high levels was 2,519,914 CPS. In tissue samples obtained from patients with low-grade bladder cancer, the average CPS value was 431,200, whereas in patients with high-grade bladder cancer, this value was measured as 447,000 CPS. These measurement values were found to be below the device's reading range of 15 ng/ml. When the CPS values of the samples were statistically analyzed, as shown in Table 1, IGF levels were found to be higher in patients with high-grade bladder cancer compared to those with low-grade tumors. According to the Independent Sample t-test we conducted, this difference was found to be statistically significant ($p<0.05$) (Table 2). As illustrated in Figure 1, which presents a box plot comparing IGF values between patients with low- and high-grade bladder cancer, IGF levels were observed to be significantly higher in patients with high-grade tumors.

Table 1. IGF-1 levels in bladder tumor grades?

Group statistics				
	Tumor	N	Mean	Std. Deviation
Igf	Low_grade_Tumor	50	43,6741	7,75930
	Hıgh_grade_Tumor	50	61,3509	11,52540

Table 2 Independent samples test

F	Sig.	t	Sig. (2tailed)	95% Confidence Interval of the Difference	
				Lower	Upper
9,287	,003	-9,015	,000	-21,61272	-13,81416

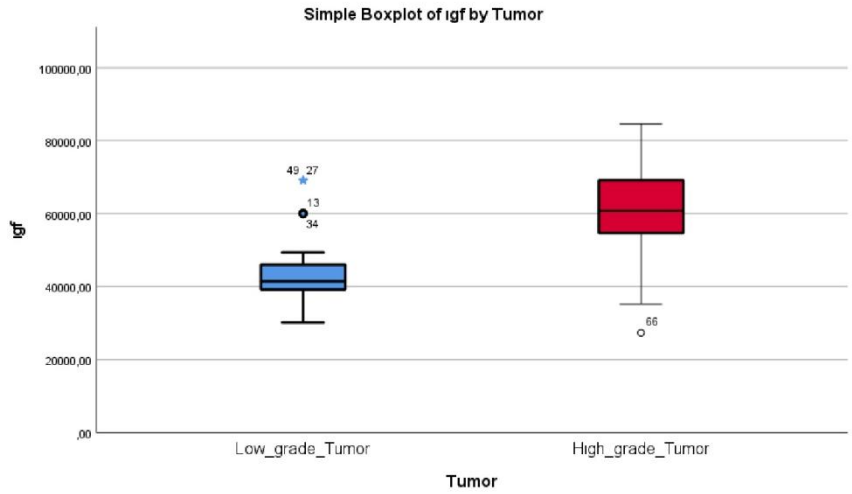

Figure 1. Low -grade and high-grade tumor box plot

Conclusion

In conclusion, this study examined IGF-1 levels in tissue samples from bladder cancer patients and found significantly higher levels in those with high-grade tumors. This finding suggests that IGF-1 may be associated with the progression and aggressiveness of bladder cancer. The proliferative and anti-apoptotic effects of IGF-1 may contribute to uncontrolled tumor growth and increased metastatic potential. However, IGF-1 levels alone do not solely determine tumor behavior; interactions with other biological factors also influence the course of the disease. Therefore, a more detailed investigation of the molecular effects of IGF-1 in bladder cancer cells is essential, and large-scale clinical studies are needed to evaluate IGF-1 as a potential prognostic biomarker.

Recommendations

Our findings indicate that IGF-1 levels are significantly elevated in high-grade bladder cancer, suggesting its potential as a biomarker of tumor aggressiveness. The ability of IGF-1 to enhance proliferation and inhibit apoptosis, particularly through PI3K/AKT and MAPK pathways, may explain the aggressive behavior of high-grade tumors. However, the study is limited by the exclusive use of tissue samples and a relatively small cohort. Future studies should include both serum and tissue analyses and investigate the IGF-1/IGF-1R axis in relation to treatment response and metastasis.

Scientific Ethics Declaration

* The authors declare that the scientific ethical and legal responsibility of this article published in EPHELS journal belongs to the authors.

*The study was approved by the Gaziantep University Clinical Research Ethics Committee (Decision No: 2024/181).

Conflict of Interest

* The authors declare that they have no conflicts of interest

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Author(s) Information

Mehmet Aydın Dagdeviren
Gaziantep University
Gaziantep University depertamant of Biyology
27000 Gaziantep/Turkiye
Contact e-mail:*aydin_dagdeviren@hotmail.com.tr*

Mehmet Ozaslan
Gaziantep University
Gaziantep University depertamant of Biyology
27000 Gaziantep/Turkiye

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