Journal of
Obesity & Weight Loss Therapy

Review Article Open Access

The Complex Link between Gastric Cancer and Obesity: Unveiling the Connection

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Abstract

Gastric cancer, also known as stomach cancer is a prevalent malignancy with significant global impact. It arises from the abnormal growth of cells in the lining of the stomach and is associated with substantial morbidity and mortality. The development of gastric cancer involves a complex interplay of genetic, environmental, and lifestyle factors. In recent years, there has been growing recognition of the role of obesity in gastric cancer development. Obesity, characterized by excessive body fat accumulation, contributes to chronic inflammation, hormonal imbalances, insulin resistance, and other metabolic dysregulations, creating a favorable environment for cancer initiation and progression. Epidemiological studies consistently demonstrate an increased risk of gastric cancer in individuals who are overweight or obese, particularly for the adenocarcinoma subtype. Adipose tissue-derived hormones, chronic inflammation, and disrupted insulin signaling pathways are proposed mechanisms linking obesity to gastric cancer. Furthermore, obesity is often associated with other risk factors such as gastroesophageal reflux disease (GERD) and unhealthy dietary habits, which further contribute to the development of gastric cancer. Understanding the complex relationship between gastric cancer and obesity is crucial for developing effective prevention and intervention strategies. Promoting healthy lifestyles, weight management, and regular medical check-ups for early detection are essential in mitigating the burden of gastric cancer and improving patient outcomes. Further research is needed to elucidate the intricate molecular mechanisms underlying this association and to explore targeted therapies that can attenuate the impact of obesity on gastric cancer development and progression.

Keywords: Gastric cancer; Obesity; Weight loss; weight management

Introduction

The obesity epidemic

Obesity is characterized by excessive accumulation of body fat, resulting from an energy imbalance between calories consumed and calories expended. The World Health Organization (WHO) estimates that over 1.9 billion adults worldwide are overweight, with more than 650 million classified as obese. Obesity is a multifactorial condition influenced by genetic, environmental, and behavioral factors. Sedentary lifestyles, high-calorie diets, and an obesogenic environment contribute to its prevalence [1-3].

Unveiling the connection

Research suggests that obesity significantly increases the risk of developing gastric cancer, particularly the adenocarcinoma subtype, which accounts for the majority of cases. Several mechanisms are proposed to explain this association

Chronic inflammation: Obesity leads to a state of chronic low-grade inflammation, characterized by elevated levels of pro-inflammatory molecules. This inflammatory milieu contributes to cellular damage and genetic alterations, promoting cancer development [4].

Insulin resistance: Obesity is closely linked to insulin resistance, a condition in which cells become less responsive to insulin, leading to elevated blood sugar levels. Insulin and insulin-like growth factor (IGF) play key roles in cell growth and division. High insulin and IGF levels may stimulate the growth of cancer cells and increase the risk of tumor formation.

Hormonal imbalances: Adipose tissue, or fat, produces hormones such as leptin and adiponectin, which regulate appetite, metabolism, and inflammation. In obesity, there is dysregulation of these hormones, potentially influencing the development of gastric cancer [5].

Gastroesophageal reflux disease (GERD): Obesity is a known risk factor for GERD, a condition characterized by the backward flow of stomach acid into the esophagus. Chronic GERD can lead to Barrett's esophagus, a precursor to esophageal adenocarcinoma, which shares some risk factors with gastric cancer.

Implications for public health: The growing prevalence of obesity worldwide poses significant challenges for public health and cancer prevention efforts. Addressing obesity not only reduces the risk of chronic diseases but may also contribute to a decline in gastric cancer cases. Public health interventions should focus on:

Health education: Raising awareness about the link between obesity and gastric cancer is crucial. Educating individuals about healthy eating habits, regular physical activity, and the importance of weight management can empower them to make informed choices.

Policy changes: Implementing policies that promote healthier food environments, limit the marketing of unhealthy products, and provide opportunities for physical activity can help tackle obesity at a societal level.

Early detection: Encouraging regular screenings for gastric cancer, especially in high-risk populations, can aid in early detection and improve survival rates.

Multidisciplinary approaches: Collaboration among healthcare

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Received: 03-Jun-2023, Manuscript No. JOWT-23-102660; **Editor assigned:** 05-Jun-2023, PreQC No. JOWT-23- 102660 (PQ); **Reviewed:** 19-Jun-2023, QC No. JOWT-23-102660; **Revised:** 23-Jun-2023, Manuscript No. JOWT-23-102660 (R); **Published:** 30-Jun-2023, DOI: 10.4172/2165-7904.1000575

Citation: Pushkar K (2023) The Complex Link between Gastric Cancer and Obesity: Unveiling the Connection. J Obes Weight Loss Ther 13: 575.

providers, researchers, policymakers, and the community is essential to develop comprehensive strategies that address the complex relationship between obesity and gastric cancer.

Literature Review

The connection between obesity and gastric cancer is an area of ongoing research, shedding light on the multifaceted mechanisms underlying this association [6]. As the obesity epidemic persists, it is crucial to recognize the potential impact on cancer rates, including gastric cancer. By adopting a multifaceted approach encompassing education, policy changes, and early detection, we can work towards reducing the burden of gastric cancer and promoting healthier lifestyles globally [6,7].

Epidemiological evidence

Epidemiological studies have consistently shown an increased risk of gastric cancer in individuals who are overweight or obese. The risk appears to be more pronounced in men and for the adenocarcinoma subtype, which is the most common type of gastric cancer. The risk tends to increase with higher body mass index (BMI) measurements [8].

Adipose tissue and cancer promotion

Adipose tissue, commonly known as fat, is not merely an inert storage depot but an active endocrine organ. It secretes various bioactive molecules, including hormones, growth factors, and inflammatory mediators. In obesity, adipose tissue undergoes significant changes, leading to chronic inflammation, altered hormone secretion, and insulin resistance, which can promote the development and progression of cancer.

Chronic inflammation

Obesity triggers a state of chronic low-grade inflammation throughout the body. Adipose tissue produces inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP). These molecules can contribute to DNA damage, cellular proliferation, and angiogenesis, creating an environment conducive to cancer initiation and growth [9].

Insulin resistance and hyperinsulinemia

Insulin resistance, a hallmark of obesity, occurs when cells become less responsive to the effects of insulin. As a compensatory mechanism, the body produces more insulin, leading to hyperinsulinemia (elevated insulin levels in the blood). Insulin and insulin-like growth factors (IGFs) have mitogenic properties, promoting cell growth and division. Higher insulin and IGF levels may stimulate the growth of cancer cells and increase the risk of tumor formation in the stomach [10].

Sex hormones and obesity

Obesity can disrupt the balance of sex hormones, such as estrogen and testosterone, leading to an increased risk of hormone-related cancers, including gastric cancer. Estrogen, in particular, has been implicated in promoting the growth of gastric cancer cells. Adipose tissue is an important site of estrogen production in postmenopausal women, and obesity can result in higher circulating estrogen levels, potentially influencing gastric cancer development.

Discussion

Obesity is a well-established risk factor for gastroesophageal reflux disease (GERD). Chronic GERD can lead to the development of Barrett's esophagus, a condition in which the lining of the esophagus undergoes cellular changes. Barrett's esophagus is a known precursor to esophageal adenocarcinoma, which shares some risk factors and anatomical proximity with gastric cancer.

Dietary factors

Obesity is closely associated with dietary habits, including the consumption of high-calorie, low-nutrient foods. Certain dietary factors, such as a high intake of processed meats, salted or smoked foods, and low fruit and vegetable consumption, have been independently linked to an increased risk of gastric cancer. It is important to note that the relationship between diet, obesity, and gastric cancer is complex and multifactorial, and further research is needed to elucidate the specific mechanisms involved [11].

Weight loss and risk reduction

Studies have shown that intentional weight loss, achieved through lifestyle modifications such as healthy eating and regular physical activity may reduce the risk of gastric cancer in obese individuals. Weight loss can improve insulin sensitivity, reduce chronic inflammation, and restore hormonal balance, thereby potentially mitigating the risk factors associated with obesity-related gastric cancer [12].

Conclusion

It is important to note that while obesity is a significant risk factor, not all individuals with obesity will develop gastric cancer. Multiple factors, including genetic predisposition, other lifestyle choices, and environmental influences, contribute to an individual's overall risk. Regular medical check-ups, screening, and adopting a healthy lifestyle are important for both cancer prevention and overall well-being.

Acknowledgement

None

Conflict of Interest

None

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