Association of Helicobacter Pylori Infection with Gastric Carcinogenesis and the Impact of Eradication Therapy

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Abstract: Globally, gastric cancer, as a highly prevalent malignant tumor, exerts great pressure on the public health system with its high morbidity and mortality rates. Studies have shown that Helicobacter pylori (Hp) infection plays a key role in the pathogenesis of gastric cancer. This Gram-negative, spiral-shaped, microaerobic bacterium is able to specifically colonize the surface of gastric mucosal epithelial cells, leading to diseases such as chronic gastritis and peptic ulcers. Prolonged chronic inflammatory stimulation may trigger pathological changes in gastric mucosal cells, such as atrophy, intestinal chemotaxis, and heterogeneous hyperplasia, which are considered to be precancerous lesions of gastric cancer. Epidemiologic studies and clinical trials have confirmed a significant association between H. pylori infection and gastric cancer, which not only significantly increases the risk of gastric cancer but also accelerates its progression. Therefore, H. pylori eradication therapy is considered a core strategy for primary prevention of gastric cancer.
***Keywords*:** Helicobacter pylori; gastric cancer; treatment

1. Introduction

Gastric cancer, as a malignant tumor with a high global prevalence, has always been at a high level of incidence and mortality. According to the global cancer statistics in 2022, the number of new cases of gastric cancer reached 968,400 and the number of deaths was as high as 659,900, which ranked fifth among the global causes of cancer incidence and death, respectively. These data fully indicate that gastric cancer poses a serious threat to human health.

2. The relationship between Helicobacter pylori infection and gastric cancer

2.1 Basic characteristics of Helicobacter pylori

Helicobacter pylori is a microorganism with unique biological characteristics, which is a micro-anaerobic, spiral-shaped, Gram-negative bacillus with flagella[1]. This bacterium is extremely demanding and is the only microorganism known to survive in the human stomach. It is able to swim freely in the gastric mucus layer and binds tightly to gastric epithelial cells via adhesins. In addition, H. pylori is able to secrete urease, which breaks down urea to produce ammonia, thus neutralizing gastric acid and creating a suitable environment for itself to survive. These unique characteristics enable H. pylori to survive and multiply in the highly acidic environment of the stomach, thereby causing damage to the gastric mucosa.

2.2 Current status and epidemiologic characteristics of H. pylori infection

Globally, the prevalence of H. pylori infection is extremely high, with approximately 50% of the population infested with this bacterium[2]. However, the prevalence of infection varies significantly between regions and populations. In developing countries, the prevalence of H. pylori infection is often higher, in some areas as high as 70% to 80%, due to factors such as poor sanitation, crowded living conditions, and contaminated water sources. In contrast, infection rates in developed countries are relatively low, ranging from 20% to 50%, largely due to better hygiene and high living standards.

Regarding the characteristics of high-risk groups, H. pylori infection and gastric cancer share common epidemiologic characteristics. The rate of H. pylori infection is high among people in areas with high incidence of gastric cancer, and the risk of gastric cancer is higher in H. pylori antibody-positive people than in negative people. In addition to regional factors, a family history of gastric cancer, poor dietary habits, poor nutritional status, and long-term alcohol stimulation are also important characteristics of high-risk groups.

2.3 Mechanisms of Helicobacter pylori infection leading to gastric cancer

H. pylori infection leads to chronic inflammation of the gastric mucosa. This inflammation is an important prerequisite for the development of gastric cancer[3]. When H. pylori enter the stomach, they adhere to the epithelial cells of the gastric mucosa, triggering an immune response and inflammatory reaction. Long-term sustained inflammatory stimulation will

gradually cause a series of pathological changes in the gastric mucosa, such as atrophy, intestinal metaplasia and so on, which are precancerous lesions for the development of gastric cancer.

H. pylori can produce many kinds of toxins and enzymes, such as vacuolating toxin, urease, etc., which have direct damaging effects on gastric mucosal cells. Vacuolar toxin can destroy the integrity of gastric mucosal cells, leading to cell vacuolization and apoptosis. Urease can break down urea to produce ammonia, which neutralizes gastric acid and creates a suitable environment for H. pylori to survive, while ammonia itself has a toxic effect on gastric mucosal cells.

H. pylori infection may also cause abnormal gastric acid secretion and changes in the microenvironment of the stomach. H. pylori infection can lead to increased or decreased gastric acid secretion, depending on the site and extent of infection. Abnormal gastric acid secretion can affect the barrier function of the gastric mucosa, making it more susceptible to damage. Meanwhile, H. pylori infection will also change the balance of microbial community in the stomach, which will favor the production and function of other carcinogenic factors such as nitrosamines.

3. Effect of Helicobacter pylori eradication therapy on gastric cancer

3.1 Principles and methods of eradication therapy

Quadruple therapy is the current mainstream program for H. pylori eradication, which combines two antibiotics, a proton pump inhibitor and a bismuth agent[4]. Antibiotics such as amoxicillin and clarithromycin can directly kill H. pylori; proton pump inhibitors such as omeprazole and lansoprazole create an environment unfavorable to the survival of bacteria by inhibiting the secretion of gastric acid; and bismuth, such as bismuth potassium citrate, helps to protect the gastric mucous membrane and enhances therapeutic effect. The drug combination and dosage of quadruple therapy need to be formulated by the doctor according to the patient's specific condition to ensure the efficacy and safety.

The treatment period of Quadruple Therapy usually lasts for 10 to 14 days, during which patients need to strictly follow the doctor's instructions and take the medication on time to avoid missing or increasing or decreasing the dosage of medication on their own. At the end of the treatment, the doctor will arrange a review, usually within 4 to 8 weeks after stopping the medication, to assess whether the H. pylori bacteria have been eradicated. Review methods include urea breath test, fecal antigen test or gastroscopy.

3.2 Role of eradication treatment in gastric cancer prevention

In terms of the effect of reducing the incidence of gastric cancer, eradication of H. pylori can significantly reduce the risk of gastric cancer. Numerous studies have shown that H. pylori infection is one of the main causative factors of gastric cancer, and about 70%-90% of gastric cancer cases are related to H. pylori infection. Through eradication treatment, H. pylori can be effectively removed from the body, thereby reducing chronic inflammation and damage to the gastric mucosa and lowering the incidence of gastric cancer. For example, a study involving thousands of subjects found that after eradication treatment, the incidence of gastric cancer significantly decreased by about 30%-50%. These data fully demonstrate the effectiveness of H. pylori eradication in preventing gastric cancer.

Eradication treatment has a significant reversal effect on precancerous lesions. Pre-cancerous lesions refer to certain diseases or lesions that have the potential to develop into malignant tumors although they are not malignant tumors per se. In the development of gastric cancer, chronic gastritis, gastric ulcer, intestinal epithelial hyperplasia, etc. are precancerous lesions. H. pylori eradication therapy not only improves the symptoms of these lesions, but also reverses their pathologic changes and reduces the potential risk of gastric cancer.

3.3 Effect of eradication treatment on the prognosis of gastric cancer patients

Eradication treatment of H. pylori can improve the therapeutic effect and survival rate of gastric cancer patients. The prognosis of gastric cancer patients is often affected by many factors, among which H. pylori infection is an important unfavorable factor[5]. Through eradication treatment, this unfavorable factor can be effectively eliminated, thus improving the therapeutic effect. Studies have shown that for gastric cancer patients, adjuvant H. pylori eradication therapy after surgical resection of the tumor can significantly improve the overall survival rate and gastric cancer-specific survival rate of patients. This improved effect is not only reflected in early gastric cancer patients, but also has positive significance for patients with progressive gastric cancer.

Eradication therapy can significantly reduce the recurrence risk of gastric cancer patients. Recurrence of gastric cancer is one of the major reasons for poor patient prognosis. Helicobacter pylori infection is closely related to the recurrence of gastric cancer, because the persistence of Helicobacter pylori will exacerbate the inflammation and damage of the gastric mucosa, which provides favorable conditions for the recurrence of gastric cancer. Through eradication treatment, H. pylori

can be effectively removed from the body, thus reducing the risk of recurrence of gastric cancer.

4. Conclusion

In summary, H. pylori eradication therapy has shown remarkable effects in gastric cancer prevention and control, which can effectively reduce the incidence of gastric cancer, improve the treatment effect and survival rate of gastric cancer patients, and reduce the risk of recurrence. However, the treatment process also faces challenges such as drug resistance, patient compliance and treatment cost, long-term effects and follow-up management. Therefore, optimizing treatment regimens, improving patient compliance, reducing treatment costs, and enhancing long-term follow-up management are key to ensuring the effectiveness and sustainability of eradication therapy.

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