Prevention of Gastric Cancer

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Gastric carcinogenesis is a multifactorial process. Chronic inflammation plays an important role in the development of gastric cancer. *Helicobacter pylori (H. pylori)* infection has been known to induce chronic gastric inflammation that leads to gastric cancer. After eradicating *H. pylori*, precancerous lesions may regress. Testing and treating for the *H. pylori* infection earlier rather than later in life is suggested to be the more beneficial approach. Fuccio L,et al. have conducted a meta-analysis of six randomized, controlled trials. This meta-analysis showed that H. pylori eradication has significant preventive effect for gastric cancer. Fukase K, et al. conducted a randomized controlled study which showed H. pylori eradication significantly reduced the development of secondary gastric cancer after endoscopic resection of primary gastric cancer. In our recent population-based study, early *H. pylori* eradication was found to be associated with decreased risk of gastric cancer.

Aspirin and non-steroidal anti-inflammatory drugs (NSAIDs) have been suggested to prevent gastric cancer by inhibiting production of COX-1 and COX-2 through both prostaglandin-dependent and independent pathways. In a meta-analysis pooling of the results of clinical studies, NSAIDs use was found to be associated with a reduced risk of gastric cancer, with similar magnitudes of risk reduction for aspirin and non-aspirin NSAIDs users. Regular users of NSAIDs were found to have a lower risk of gastric cancer compared to nonusers and irregular users. Another meta-analysis also reported similar protective effect of NSAIDs in gastric cancer. However, the risk of cardiovascular events and gastrointestinal bleeding associated with NSAIDs and COX-2 inhibitors outweighs their beneficial effects in the chemoprevention of cancer in average-risk individuals.

High intakes of fresh fruits and vegetables have been shown to be inversely associated with risks of gastric cancer. One randomized trial has shown that subjects who received factor D (a combination of selenium, vitamin E, and beta-carotene) had lower gastric cancer mortality in a poorly nourished population in Linxian. However, a meta-analysis consisting of 14 randomized control trials have failed to find a protective effect of Vitamin A, Vitamin C, Vitamin E, β-carotene, riboflavin, and zinc, either single use or combinations of them. Only selenium showed a reduction on the incidence of gastrointestinal cancers in the sub-analysis of four trials. Collectively, there is still insufficient data to support the beneficial effect of anti-oxidant vitamins supplement for well nourished subjects in the prevention of gastric cancer.