Lifestyles and their Close Relationship with Gastrointestinal Diseases, Part II: Smoking, Obesity, Exercise and Alcohol

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Abstract: The gastrointestinal (GI) tract is responsible for ingestion of food and beverages, its propulsion, digestion, absorption, and finally excretion of its unabsorbed waste products. It is one continuous tube, measuring about 7–11 meters. Different sections have different structures, pH, and functions. Symptoms of a GI disease may differ according to the location and type of lesion. In the oral cavity, the patient may have trouble eating and swallowing. Esophageal reflux may cause heartburn, peptic ulcer disease and pancreatitis may cause stomach pain, while ailments of the intestines may cause malabsorption and diarrhea. GI ailments can greatly reduce the quality of life. They can also hasten mortality. The GI system is strongly affected by lifestyle factors, with unhealthy lifestyles increasing the risk and progression of various GI ailments. This manuscript looks at its relationship with four lifestyle factors, namely smoking, alcohol intake, exercise, and obesity. Its relationship with diet was discussed in part I of this two-part manuscript.

Keywords: Gastrointestinal diseases, smoking, exercise, alcohol, obesity, lifestyles.

INTRODUCTION

The gastrointestinal (GI) tract (alimentary canal or gut) is an extensive tubular track (about 7–11 meters long) consisting of the oral cavity, pharynx, esophagus, stomach, small intestine, large intestine, and ending in the anus [1]. The accessory glandular organs include salivary glands, liver, gallbladder, and pancreas. The main functions are ingestion, propulsion, digestion, absorption, and excretion of waste products [2]. Gastrointestinal diseases affect the gastrointestinal (GI) tract from the mouth to the anus. The major GI diseases are oral lesions [3], esophagitis [4], esophageal reflux [5], peptic ulcer [6], irritable bowel syndrome [7], inflammatory bowel disease [8], and GI cancers [9]. They commonly manifest as oral discomfort, heartburn, abdominal pain, abdominal distention, gastrointestinal bleeding, intestinal obstruction, malabsorption, malnutrition, and diarrhea [10]. The GI tract is extremely susceptible to lifestyles. This manuscript limits the discussion to the common GI diseases. Hepatic disorders and their relationship with lifestyles have been discussed in a separate publication [11]. Part I of this two-part manuscript discussed the role of diet on gastrointestinal diseases. Part II of this manuscript discusses the role of four other lifestyle factors, namely smoking, obesity, alcohol consumption, and exercise, and their impact on gastrointestinal diseases.

DISCUSSION

Diseases depend on several factors, which include genetics, environmental and social factors [12]. Lifestyles are increasingly becoming known as a modifiable risk factor for most chronic diseases. The five most impactful lifestyles are smoking, alcohol intake, obesity, exercise, and diet [13]. Healthy lifestyles not only help prevent most non-communicable diseases but also help mitigate their progression and reduce mortality [14]. Li et al. in a recent study from Harvard University in the USA reported that at age 50, adopting all five healthy lifestyles can potentially provide another 43.1 years of life in females and 37.6 years in males [5].

Smoking

The upper aerodigestive tract is the first to be exposed to the gaseous and particulate chemicals present in tobacco smoke (cigarette, cigar, or water pipe). Besides the damage to the dental structures, it causes inflammation of the oral cavity, increases susceptibility to candida infections, may induce premalignant lesions, and increase the tendency to develop oral, laryngeal, and pharyngeal cancer [16, 17]. Vaping is also associated with increased development of oral ulceration [18]. It also increases the incidence of esophagitis [19]. It weakens the esophageal valve,
encouraging reflux esophagitis and gastroesophageal reflux disease (GERD). Since bile salts tend to move more rapidly from the intestine to the stomach in smokers, and by directly injuring the esophageal mucosa, the reflux in smokers is more serious [20]. The reflux events are also increased in smokers [21]. Several studies have shown that cigarette smoking increases the incidence of peptic ulcers and their progression [22, 23]. Besides decreasing the bicarbonate production by the pancreas and stimulating the retrograde influx of bile acids into the stomach, smoking also interferes with antioxidant and immunity activity protecting the gastric mucosa [24]. The amount of nicotine in the gastric fluid is 10 times higher than in the arterial blood and 80 times higher than in venous blood [25]. Smoking also increases the secretion of gastric acid and lowers the stomach pH [20]. Studies show that the esophagus in smokers is subject to a greater acid exposure time on ambulatory pH monitoring [26]. Smoke also has vasoconstrictor and procoagulant effects, deleteriously affecting gastric microcirculation [27]. These changes contribute to the increased susceptibility to H. Pylori [28] – the main cause of peptic ulcer disease [29]. H. pylori infiltraion has been found to be denser in the gastric antrum of smokers [30]. These mechanisms also increase the failure rate of ulcer healing [22] and encourage relapse [31]. The risk of H. pylori eradication failure in smokers increases with the continuation of smoking and a high smoking dose [32]. The effects of smoking on duodenal ulcers are unclear. Smoking increases gastric emptying rates and disturbs duodenal prostaglandins. However, no causal effect of smoking was noted, when investigated prospectively in more than 47,000 men with duodenal ulcers [33]. Tobacco smoking increases the risk of acute (AP) and chronic (CP) pancreatitis and these two combined [34]. There is a dose-response relationship between the increasing number of cigarettes and pack-years smoked and pancreatitis risk. A systemic review and meta-analysis of ten studies revealed that the summary risk ratio (RR) for acute pancreatitis was 1.49, for current smokers, 1.24. For former-smokers and 1.39 for ever-smokers compared to never-smokers [34]. The two main metabolites from cigarette smoke, namely nicotine and NNK 4-(methylnitrosamino)-1-(3-pyridyl)-1- butanone are acutely harmful to the pancreas, as seen in cases of AP [35].

Self-reported irritable bowel syndrome (IBS) in the general population shows a strong association with smoking [36]. Cigarette smoking also significantly increases the risk of Crohn’s disease [37]. These patients also experience poor outcomes. They are more likely to develop complications, are more likely to get hospitalized, have a worse response to treatment, and need surgery more often [38]. Ironically, epidemiological observations indicate that cigarette smoking confers some protection against ulcerative colitis [39, 40]. A meta-analysis found an odds ratio of 0.58 in smokers for the development of ulcerative colitis [41]. As a matter of fact, nicotine has been tried therapeutically in these patients [42, 43]. Besides the increased risk of oral, pharyngeal, and laryngeal cancer [44-46], smoking also increases the incidence of cancer of the esophagus, mainly due to the higher rates of Barret’s esophagus seen in this population [47,48]. An increased risk for gastric cardia and other stomach/intestinal cancers has also been noted in smokers [49]. It also increases the risk of colorectal polyps [50] and colorectal cancer [51]. These patients have a poorer prognosis when compared to non-smokers [52]. Smoking cessation is associated with reductions in cancer [53, 54]. And finally, smoking may also be linked with a higher risk of developing gallstones [55].

Obesity

According to World Health Organization (WHO) in 2016, based on a body mass index (BMI) of ≥25 Kg/m², more than 1.9 billion adults were overweight, and based on a BMI based of ≥30 Kg/m², almost 650 million people were obese [56]. Overweight and obesity are well-known risk factors for a variety of GI disorders. It is established to be a strong risk factor for reflux esophagitis and Barrett’s esophagus [57, 58]. The association of esophagitis with overweight has an odds ratio of 1.33, and with obesity, an odds ratio of 1.70 [59]. Increased intra-abdominal fat induces the reflux of the stomach contents into the esophagus. Obesity has been reported as a risk factor of peptic ulcer disease (PUD) in many studies [60-63]. However, Tsai et al. reported conflicting data [64]. In a recent study on 32,472 individuals without PUD at baseline, Pyo et al. found a significantly higher cumulative incidence of PUD in obese subjects compared to non-obese subjects [65]. However, when adjusted for possible confounding factors, the association was no more significant. Overall, the association between obesity and PUD remains inconclusive. Overweight and obese patients have a higher incidence of biliary disease [66, 67] and pancreatitis [67]. The biliary disease causes acute pancreatitis by stones, sludge, or micro-lithiasis in the bilio pancreatic passages, either by causing bile reflux or increasing pancreatic duct pressure [68]. Obesity is also associated with hypertriglyceridemia (HTG) [69]. Obesity can unmask primary HTG from genetic causes [70] and is a risk factor for secondary HTG [71]. Obesity is also associated with type 2 diabetes mellitus (T2DM) which can increase the risk of pancreatitis by increasing triglycerides [69], cholelithiasis [72], or beta cell hypertrophy [73]. Certain surgical interventions to treat obesity may also increase the risk of pancreatitis [74, 75]. These include Roux-en-Y gastric bypass surgery [76], duodeno-jenunal bypass [77], and gastric balloon insertion [78]. Obesity is also associated with colonic diverticulosis [79]. In an endoscopy-based prospective study, the multivariable-adjusted odd ratios for diverticulosis were 3.02 in individuals with a BMI of 25.0–29.9 kg/m2 and 4.43 in individuals with a BMI of ≥30 Kg/m2.
of 30.0 kg/m2 or greater [80]. The disease is also more complicated during its course in obese individuals [81]. Data from the scientific literature have failed to show a possible association between obesity and the incidence of irritable bowel syndrome (IBS) [82]. However, those with abdominal obesity appear to have more frequent symptoms [83]. Obese individuals have a higher risk of inflammatory bowel disease (IBD) [84], especially Crohn’s disease (CD) when compared to ulcerative colitis (UC) [85]. Visceral obesity, when associated with IBD is more harmful – patients with CD have a higher probability of surgery and of penetrating disease while those with ulcerative colitis are at an increased risk of relapse [86]. Obesity is also associated with an increased risk of several GI cancers, including esophageal adenocarcinoma, proximal gastric carcinoma, pancreatic cancer, and colorectal cancer [87]. Islami et al. reported that in the USA, in 2014, extra body weight accounted for 32% of esophageal, 17.5% of gastric, and 17% of pancreatic cancers in adults aged 30 years and older [88]. A meta-analysis showed that an increase of 5 kg/m2 in BMI in men correlates with a relative risk of 1.24 for colon cancer [89]. A causal association between obesity and different types of GI cancer is well documented. Moreover, excess weight is also well-known as a risk factor for cancer mortality [90]. Cancer is enhanced by obesity primarily due to increased inflammation and increased insulin resistance [91, 92].

**Alcohol**

Several studies have shown that alcohol consumption is a trigger factor for GERD [93-95]. Heavy drinking also appears to be associated with PUD. Anderson et al. reported that drinking more than 42 drinks per week increased the risk of a bleeding ulcer fourfold (Risk Ratio=4.4) when compared with drinking less than one drink per week. On the other hand, several large-scale prospective studies have suggested a protective effect of moderate alcohol consumption (one or two drinks a day) on the development of gastric ulcer [97, 98]. Some studies report that wine drinkers have a reduced risk of ulceration, while spirit drinkers have an increased risk [99]. It is unclear if alcohol intake increases duodenal ulcers. No causal association was found in a prospective study of more than 47,000 men [100]. Alcohol is a primary cause of both AP and CP in most developed countries [101]. Chronic alcohol consumption causes 17% to 25% of AP cases worldwide and is the second most common cause of AP after gallstones. It is estimated that globally, 40% to 70% of CP is alcohol induced [102]. Chronic abusers of alcohol with pancreatitis have a poor prognosis [103].

There is limited data available on the role of alcohol in IBS [104]. Studies seem to indicate that alcohol consumption in low to moderate amounts has no demonstrable effect on IBS [105, 106]. However, heavy intake appears to increase symptoms associated with IBS [107]. The evidence linking the consumption of alcoholic beverages and the development of new-onset IBD is not clear [108]. Zutshi et al. found no effect of alcohol intake on IBD [109]. Hsu et al., on the other hand, reported that the risk of IBD was higher in patients with alcohol intoxication [110]. It has however been noted that IBD patients experience more gastrointestinal symptoms following alcohol consumption [111]. Further, alcohol use appears to increase the risk of relapse [112]. The use of alcohol can also interfere with several medications used to treat IBD (such as mesalamine, azathioprine, methotrexate, and biologic drugs), leading to increased adverse events or even loss of efficacy [113,114]. Alcohol consumption also increases the risk of several GI cancers. Yoo et al. in a study of 319,202 individuals found that compared with nondrinkers, the risk for GI cancer was elevated in all alcohol drinkers - mild drinkers (adjusted hazard ratio [aHR], 1.04), moderate drinkers (aHR, 1.14), and heavy drinkers (aHR, 1.28) [115]. These include cancers of the upper aerodigestive tract (including the esophagus), stomach, colon-rectum and pancreas [116-118]. Alcohol intake is also detrimental to lower GI tract cancer. According to the National Cancer Institute, there is a 1.2-to-1.5-fold increased risk of cancer of the colon and the rectum in alcohol drinkers [119].

**Exercise**

Exercise, especially in athletes. Appears to lead to an increase in the frequency and duration of GERD [120]. The relationship appears to be dependent on the intensity of exercise [121]. Runners frequently report heartburn/reflux [122]. Postulated mechanisms for this increase may include decreased GI blood flow, alterations in hormone secretion. Changes in the motor function of the esophagus, and the constrained body position during exercise. However, in a recent systemic review of 72 articles, Zhang et al. concluded that exercise (physical exercise >30 minutes (>3 times/week) was in general beneficial (odds ratio >3) for GERD [123]. It appears that although moderate degree of exercise is beneficial, higher intensity of exercise may lead to a worsening of GERD [124]. Physical exercise appears to have a similar association with PUD. A moderate amount of exercise has a favorable impact on several risk factors for peptic ulceration, including a reduction in gastric secretions, enhanced immunity, and a reduction in stress [125, 126]. Results suggest that the benefits may be more with duodenal ulcer, with regular exercise helping in the healing and maintenance of remission [127-130]. Further, regular physical activity is associated with a decreased risk for severe GI hemorrhage in older subjects with gastroduodenal ulcers or gastritis (RR = 0.4) [131]. Low-intensity exercise also appears to help both IBS and IBD [132-134]. As noted with GERD and PUD, GI symptoms may be aggravated with increasing intensity and duration of the exercise [135,136]. There is strong evidence that physical activity of moderate to vigorous intensity protects against several GI cancers.
In a large study (1.44 million people, ages 19 to 98) Moore et al. examined several cancers and concluded that leisure-time physical activity was associated with a significantly decreased risk of esophageal adenocarcinoma, gastric cardia cancer, and colon cancer [137]. Pre-rehabilitation exercise helps decrease postoperative complications in GI cancer patients [138] and is associated with long-term benefits [139]. Exercise in GI cancer patients also helps reduce fatigue and improve functional capacity [140]. The quality of life is also improved during cancer treatment [141]. Overall, exercise during all phases of GI cancer helps improve outcomes in these patients [142, 143].

CONCLUSIONS

Healthy lifestyles can dramatically reduce the development and progression of major gastrointestinal diseases. These include GERD, PUD, IP and CP, IBS and IBD, and several GI cancers. Smoking, whether active or passive, is invariably harmful (except maybe for UC). Low to moderate alcohol intake and exercise is helpful for many GI conditions. In excessive amounts, however, both alcohol intake and exercise may cause harm. Obesity worsens all GI diseases, including increasing the risk of several cancers. And finally, a prudent diet, as discussed in part I of this manuscript, is beneficial. Certain dietary components may aggravate GI disorders and should be avoided depending upon the nature of the disease, and the experience of the patient. Overall, the five lifestyles discussed in this two-part manuscript, if followed in a healthy way, greatly help reduce GI morbidity and mortality.

REFERENCES


