Alcohol and its effects on the gastrointestinal tract

Alcohol has many deleterious effects on the gastrointestinal tract, including impaired gut motility, mucosal damage and carcinogenesis, write Grace Chan and Claire Smyth

IT IS REPORTED THAT IN 2010, the average Irish person above the age of 15 drank 11.9 litres of pure alcohol in a year. This is equivalent to approximately 44 bottles of vodka, 470 pints of beer or 124 bottles of wine. The effects of alcohol in chronic liver disease and liver-related portal hypertension have been widely described including the development of oesophageal and rectal varices, portal hypertensive gastropathy and gastric antral vascular ectasia (GAVE) syndrome. However, the direct effects of alcohol on the gastrointestinal tract are less well-known.

Given the increasing quantity of alcohol consumption in this country, it is not surprising that a considerable number of referrals to the average gastroenterology clinic and to casualty can be partially attributed to alcohol.

Alcohol is generally divided into three types: wine, beers and liquors, and despite alcohol being a common ingredient in all three, some beverages contain substances that may exert additional effects on the gastrointestinal tract.

Oropharynx and oesophagus

The oropharynx and oesophagus is exposed to undiluted alcohol immediately upon consumption. As a result, mucosal injuries occur quite frequently in people who consume large amounts of alcohol. In combination with poor nutritional status, glossitis and stomatitis may result. There is also an increased incidence of tooth decay, gum disease and loss of teeth among alcoholics. Chronic alcohol abuse also damages the salivary gland, thereby interfering with saliva secretion. In alcoholics, this results in parotid hypertrophy.

In the oesophagus, alcohol reduces the pressure of the lower oesophageal sphincter and causes peristaltic dysfunction. There is an increase in the amplitude of the contractions in the middle third of the oesophagus and a decrease in the lower/middle amplitude ratio, thereby increasing gastro-oesophageal reflux disease (GORD). Reflux symptoms are further precipitated by the decrease in salivary bicarbonate and the peripheral neuropathy affecting muscle contraction and acid clearance from the oesophagus. However, the evidence for the relationship between heavy alcohol consumption and GORD has not been consistent. In Mallory-Weiss syndrome, which is characterised by mucosal tears around the gastro-oesophageal junction following retching or vomiting, excessive alcohol consumption is held accountable in up to 60% of cases.

The effects of alcohol on the oesophagus extend beyond the direct damage by alterations in the epithelial transport, intracellular junction disorders and impairment of the mucosal barrier. Alcohol sensitises the mucosa to further
damage caused by the penetration of hydrogen ions into the mucosa.\textsuperscript{12}

**Stomach**

Chari et al previously conducted a review which found that low-dose alcoholic beverages such as white and red wine were found to stimulate acid secretion and gastrin release.\textsuperscript{13} However, high-grade alcoholic beverages such as whiskey, gin and brandy did not.\textsuperscript{13} The proposed mechanism for the stimulatory effect of low-dose alcohol includes mediation via the cholinergic system, topical stimulation of the parietal cells with an increase in cyclic AMP production and histamine release.\textsuperscript{14}

Fermented and non-distilled alcoholic beverages (eg, beer, wine, champagne) have also been shown to increase acid and gastrin secretion to a greater extent compared to distilled beverages (eg, whiskey, brandy), independent of the total alcohol content.\textsuperscript{15}

Alcoholics are more prone to gastric mucosal inflammation, with approximately 25% with those enrolled in a detoxification programme suffering with superficial gastritis and a further 24% with chronic atrophic gastritis. This compares to 11% and 3% respectively in healthy subjects.\textsuperscript{16} However, this increase in mucosal inflammation has not been shown to translate into an increased incidence of peptic ulcer formation or reduction in ulcer healing.\textsuperscript{17}

The dose of alcohol also appears to have an effect on gastric emptying. Low-dose alcohol has been shown to increase gastric emptying and initiate pyloric relaxation.\textsuperscript{18,19} Conversely, beverages with high alcohol concentrations delay gastric emptying, thereby increasing the effects of bacterial degradation of food, resulting in bloating and fullness;\textsuperscript{4} a common complaint in patients with functional disorders. There is also evidence to demonstrate that alcohol consumption increases the likelihood of functional dyspepsia and recurrent abdominal pain.\textsuperscript{20}

**Intestine**

Alcohol is rapidly absorbed in the small intestine. Previous studies demonstrate that alcohol concentration can drop from 10% to less than 1.5% over a distance of 30cm in the jejunum.\textsuperscript{21} The most common intestinal disorders encountered by alcoholics are diarrhoea and malnutrition.

Diarrhoea is usually a result of a combination of factors. Firstly, chronic alcohol consumption leads to markedly reduced water and sodium absorption in the jejunum and ileum, resulting in diarrhoea.\textsuperscript{22} Steatorrhea is also known to occur in alcoholics with pancreatic insufficiency and alcoholic liver disease. Nevertheless, increased faecal fat and nitrogen excretion has also been reported in alcoholics without cirrhosis or chronic pancreatitis.\textsuperscript{11,23} The latter may be partially explained by the finding that ethanol interferes with the intestinal hydrolysis of peptides.\textsuperscript{24}

Acute exposure of the small intestinal mucosa to alcohol appears to inhibit the active transport of numerous nutrients across the epithelial layer.\textsuperscript{25} In the longer term, alcohol affects the absorption of monosaccharides such as D-xylose\textsuperscript{26}, folic acid,\textsuperscript{26} B12,\textsuperscript{27} calcium\textsuperscript{28} and vitamin C.\textsuperscript{29}

Despite the normal appearances of the intestinal mucosa in alcoholics under light-microscopy, structural changes have been noted when quantitative morphometric methods or electron-microscopical examinations were carried out. These changes include reduced villus height, reduced mucosal surface area of villi, increased intra-epithelial mononuclear cells, goblet cell hyperplasia and gastric metaplasia.\textsuperscript{30,32} The increased number of myofibroblast-like cells in the duodenal mucosa of alcoholics suggest the development of fibrosis of the intestinal mucosa.\textsuperscript{21} However, most of the malnutrition associated with alcohol is related to deficient dietary intake rather than actual malabsorption and most are correctable with nutrient replacement.

In contrast to the upper gastrointestinal tract, the colon is exposed only to alcohol concentrations similar to that in the bloodstream. In healthy humans, alcohol administration has been shown to reduce the frequency and strength of muscle contraction in the rectum and may potentially reduce the faecal transit time.

**Carcinogenesis**

The World Health Organization (WHO) lists acetaldehyde associated with alcoholic beverages as a Group 1 carcinogen. In the gastrointestinal tract, alcohol consumption has been linked to an increased risk of tumours of the oral cavity, pharynx, oesophagus and stomach, particularly in the presence of tobacco exposure.\textsuperscript{33,35}

In an epidemiological study, it was found that people who consumed more than 21 beers or liquors a week had a relative risk of 5.2 of developing oropharyngeal and oesophageal tumours, whereas those subjects in this group who included wine in their consumption presented a decrease in relative risk to 1.7.\textsuperscript{34} Similarly, moderate consumption of liquors and beers but not wine, has been linked to an increased risk of colorectal carcinoma and adenomas.\textsuperscript{35,36}

Acetaldehyde is a by-product of alcohol production and its concentration depends on yeast used, fermentation process, distillation and preservation. High acetaldehyde concentration in Calvados (French apple brandy) has been suggested to partially explain the high incidence of oesophageal cancer in some regions in France.\textsuperscript{37}

Similarly, high concentrations of acetaldehyde (\(> 1,000\text{mg/l}\)) in sweet spirits are thought to be partially responsible for the high incidence of oral and oesophageal cancer in Eastern Europe.\textsuperscript{38} This level is more than 200 times higher than the acetaldehyde concentration known to be able to produce mutagenic changes in DNA.\textsuperscript{39}

In individuals with aldehyde dehydrogenase (ALDH2) gene mutations, acetaldehyde concentrations in the saliva appear to be two to three times higher than in those with normal ALDH2 genotype.\textsuperscript{40} These individuals have been shown to be more susceptible to upper gastrointestinal cancers due to the enhanced exposure to acetaldehyde.\textsuperscript{41}

**Conclusion**

Alcohol has many deleterious effects on the gastrointestinal tract, distinct from the well-known and described effects on the liver and the consequences of portal hypertension. These include effects on gut motility, absorption, mucosal damage and carcinogenesis. The rise in alcohol consumption in the recent years has lead to a greater need for awareness among all physicians to ensure accurate diagnosis and treatment of alcohol-related gastrointestinal disorders.

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