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ALCOHOLISM ASSOCIATED GASTRITIS: AN OVERVIEW

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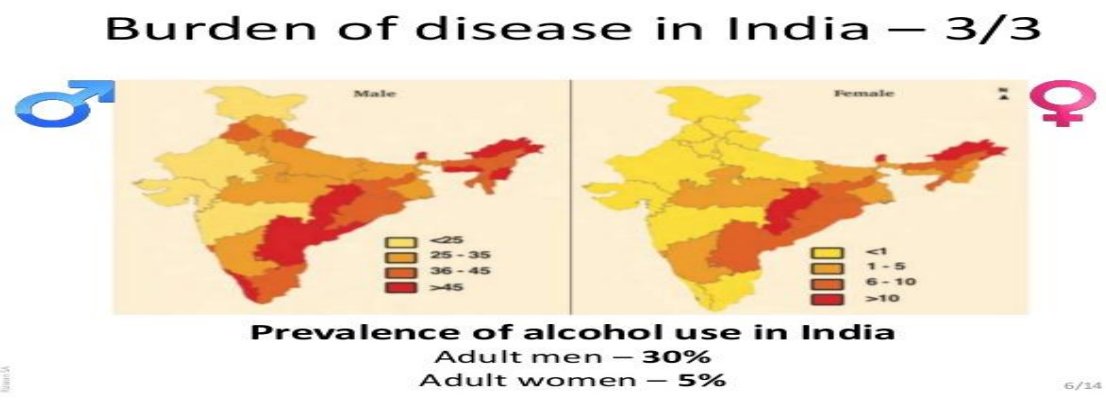
INTRODUCTION

Alcohol is a psychoactive substance and is widely used in many cultures for centuries, its harmful uses may leads to large diseases, social and economic crisis in societies. Alcoholism is generally characterized as a family disease in the sense that it affects everyone in the family in all aspects—socio-economically, spiritually, physically, psychologically, and emotionally. Families and homes of alcoholics suffer much from the degradation and stigmatization attached to the individual alcoholic. To some extent, this may reflect the negative perception held by society about alcoholism. The relational and communal nature of our cultural thinking and practice also emphasizes, as in Family systems theory, the fact that whatever affects the individual member of the family affects the whole family and also the community. Alcoholics required to be integrated into the mainstream society by providing suitable rehabilitation. In the due course of rehabilitation, alcoholics suffer from severe withdrawal symptoms and gastritis. Alcohol is a cause of chronic gastritis and the duration of excessive drinking is proportional to the severity of the mucosal lesion. The effect of alcohol on the gastric mucosa is not mediated by malnutrition, hepatic damage, intestinal malabsorption, anaemia, ascorbic acid deficiency, or any disturbance in immune tolerance but direct one. Alcohol consumption leads to negative consequence for the drinker, to his immediate environment and the society. Social problems such as traffic accidents, workplace related issues, domestic violence and interpersonal misdemeanour have been the goals of research attention in the current era. The human body is adversely affect when heavy amount of alcohol is ingested and various organs

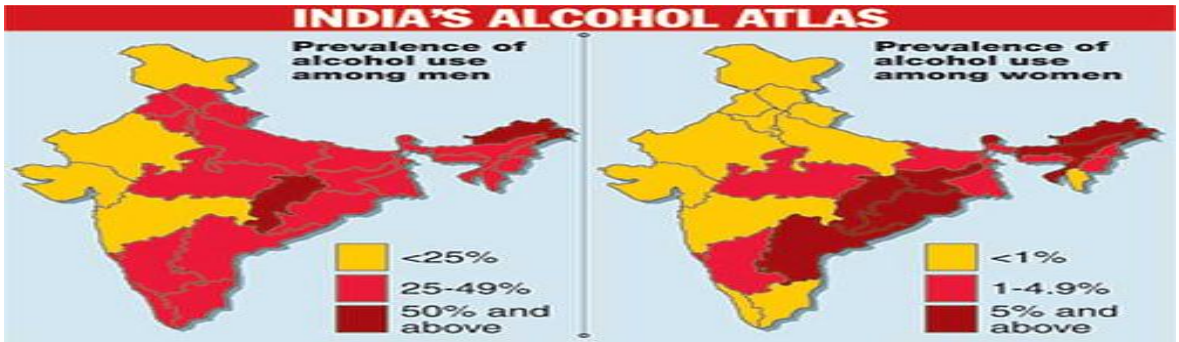
get deleterious effect mainly to the brain and gastrointestinal system by the alcohol metabolite, acetaldehyde.[1] Alcohol can decrease lower oesophageal sphincter pressure, inhibit the peristalsis of the distal oesophagus, and alter functioning of parotid gland and its secretion.[2] It is the major cause of both acute and chronic pancreatitis. A study in– vivo and in-vitro (using pancreatic tissue from patients with alcohol-induced chronic pancreatitis and from animal models of experimental pancreatitis) indicate pancreatic stellate cells (psc) are activated by alcohol exposure and leads to pancreatic fibrosis by mitogen – activated protein kinase pathway.[3,4,5] According to world Health Organization, Development of spectrum of liver disease, including hepatic steotosis, alcoholic hepatitis, alcoholic fibrosis, cirrhosis and hepato cellular damage are caused due to excessive alcohol consumption.[6,7] Recent studies reveals the strategic role of alcohol in gastric carcinoma and colon cancer and possibility of immune suppression action of alcohol and augment the magnitude of free radical.[8] Ultimately contribution to gastritis by alcohol, which irritates the stomach[9] in which inflated production of gastric acid can be seen, which can leads to peptic ulcers and potential bleeding.[10]

Epidiemiology: World market globalisation, and economic development, the incidence of alcohol related health and social problems are expected to rise and eventually increases with alcohol consumption. According to who global status worldwide consumption in 2010 was equal to 6.2 litres of pure alcohol consumed per person aged 15 years or older and this excessive alcohol consumption is considered as worldwide major health issue, which accounts for nearly 1.8 million deaths per year (3.2% of all deaths).[11] In india, undocumented alcohol consumption and sale is increased by nearly three times between 2000 and 2009 and is examined on heavy alcohol use within a community can guide policy develop related to alcohol availability and sale. Large number of alcohol abstainers can be shown in studies even India is `dry`nation.[13]



Based on The National Household Survey on Drug Abuse in India (2001) report 74.1% of males were life time alcohol abstainers and during the previous month 21.4% had used alcohol and estimated that India had 62.46 million alcohol users while roughly only one sixth (11.08 million) used drugs other than alcohol.[14] The National Family Health Survey - 3 (NFHS 3) reported that men aged between 15 to 54 years of 32% while 2% women only consume alcohol.[15] Despite the fact that a large proportion of the Indian populace are abstainers, the pattern of alcohol consumption among those who drink is worrying. Research has shown that more than 50% of alcohol consumers satisfy the criteria for hazardous drinking in India.[16] Signature pattern in India was one of heavy drinking wherein more than five drinks are consumed on a typical occasion.[17] More than wine and beer indians prefer more to distiled spirits with high percentage of alcohol and Alcohol sales in Tamil Nadu reported that production of distilled spirits was higher than beers and its about 100 million litres.[16]

The 2011 NIMHANS reported that among the educationally deprived populations, alcohol use is more prevalent. Analysing household expenditure, a Government report showed that regular alcohol use was more among those below the poverty line (5.9%) when compared to others (3.9%) with a ratio of 1.5.[13] Another study showed that while 12% of men with low-est wealth quintile aand 6% of men in the highest wealth quintile drank almost daily.[15] Examining alcohol availability and use in a rural area in Tamil Nadu, a study summarised that use of Indian-made foreign liquor (distilled spirits) and living in a village which brewed illicit alcohol increased the risk for hazardous use.[18] This indicated that easy access to cheap and strong liquor facilitates the increased risk related to alcohol use.



Prevalence: Only small number of studies conducted on smaller population in different regions of India. In northern India, the alcohol use found to be between 25% and 40% in one year prevalence and in southern India it is between 33% and 50% and the poor and lesser

educated shows lesser prevalence among the people.[17,19] In North Indian rural population, the alcohol misuse is found to be 11.3%among 55-64 years and 16.8% among 65-74 years age group.[18] Another study reveals about the current use and ever use of alcohol among those of >50 years of age and it is found out as 18.3% and 23.3% respectively.[19] A very high rate is reported in a Sikkim study when compared to national average and it is about 35% of the population >21 years are chronic alcoholic and also it shows a very high relapse rates after de-addiction for alcohol abuse. However those findings are need to interpreted with caution because those are small samples.[20] In the community of western India, a study conducted among 50 220 middle aged people and found the 18.8% are currently using alcohol and 4.9% are past users and 76% are not ever used alcohol and the prevalence of current use decreased in the higher age groups(21.5% in the 55-59 years age group to 5.7% in the years of >85 years age group). The ever prevalence over 40% subjects among who speak kannada, malayalam, tamil,and telungu speaking persons are noted while its only 15% or less among Gujarati, hindi ,urdu speaking persons. The highest prevalence for ever use was found on christians(61.2%) and least to Muslims (9.4%) and likewise the prevalence was high among primary educated (27.1%) followed by illiterates (25.6%).[21]

Parameters	*n*	Percentage (%)
Age		
18-30	86	23
31-45	167	44
46-60	125	33
Education		
Graduate	21	5
Primary	212	56
Secondary	65	17
Illiterate	80	22
Employment status		
Labour	40	10.58
Professional	17	4.49
Semiskilled	59	15.60
Skilled	26	6.87
Studying	12	3.17
Unemployed	224	59.25
Family History		
Yes	275	72.75
No	103	27.24
Living Circumstances		
Alone	80	21.16
With family	285	75.39
With Friends	13	3.4
Economic Status		
Lower	231	61
Middle	129	34
Upper	18	5

In Tamil Nadu, alcohol consumption across the districts shows that in most of the districts, thelevel is less than 10 percent (Table 7.4). The prevalence of alcohol use across the state ranges from 6 percent in Chennai, Ariyalur, Theni and Virudhunagar to about 14 percent in Sivaganga. Consumption of alcohol is found high in districts such as Khancheepuram (12%), Karur (13%) and Tiruppur (around 14%). Other districts with more than 10 percent of adults consuming alcohol are Namakkal, Cuddalore, and Madurai.

Table 7.4 PERSONAL HABITS

Percentage of all persons (age 15 years and above) classified as having personal habits by districts, Tamil Nadu, 2012-13.

Districts	Percentage of all persons			Total number of all persons covered**
	Using Smokeless Tobacco	Smoking	Consuming Alcohol	
Thiruvallur	4.8	5.5	8.9	3870
Chennai	2.1	4.1	5.9	3913
Khancheepuram	9.2	6.7	12.0	3439
Vellore	4.6	5.6	8.2	5075
Dharmapuri	6.1	8.5	9.7	4718
Tiruvannamalai	4.5	6.2	8.9	3359
Viluppuram	11.6	6.5	9.9	3751
Salem	11.2	8.0	7.8	3737
Namakkal	8.4	8.2	10.3	3920
Erode	13.6	10.1	9.6	3504
Nilgiris	7.9	8.0	8.8	3142
Coimbatore	11.2	9.2	9.2	3703
Dindigul	11.8	8.1	9.4	3046
Karur	10.2	11.4	13.1	3720
Tiruchirappalli	7.7	7.4	8.4	2807
Perambalur	12.0	5.5	9.0	3262
Ariyalur	12.5	3.5	5.5	3202
Cuddalore	8.5	6.8	10.2	3476
Nagapattinam	16.5	7.4	9.8	4403
Thiruvarur	16.0	7.2	9.6	4169
Thanjavur	14.5	7.4	8.7	3696
Pudukkottai	10.6	5.1	7.4	3375
Sivaganga	14.0	8.7	14.1	4303
Madurai	6.5	9.2	10.9	2405
Theni	4.1	6.4	5.6	3460
Virudhunagar	4.3	5.1	5.8	3491
Ramanathapuram	3.1	4.9	7.8	4576
Thoothukkudi	3.7	6.4	8.5	3366
Thirunelveli	3.6	6.9	6.9	3205
Kanniyakumari	4.3	5.7	7.2	3332
Krishnagiri	10.2	7.8	7.3	5154
Tiruppur	12.7	12.3	13.5	3349
DLHS-4	8.8	7.2	9.0	117928

**Unweighted cases.

Source: TAMIL NADU DISTRICT LEVEL HOUSEHOLD AND FACILITY SURVEY (2012-13)

PATHOPHYSIOLOGY OF GASTRITIS IN ALCOHOLICS

When Alcohol is ingested it reaches into blood stream via stomach walls after passing through oesophagus.[22] Alcohol induces gastritis by the production of more amount of gastric acid and also along with peptic ulcer and potential bleeding. ICD-10 code bearing the number K 29.2 is about alcoholic gastritis.[23] Moderate drinking may cause acute and heavy drinking leads to chronic gastritis.[24]

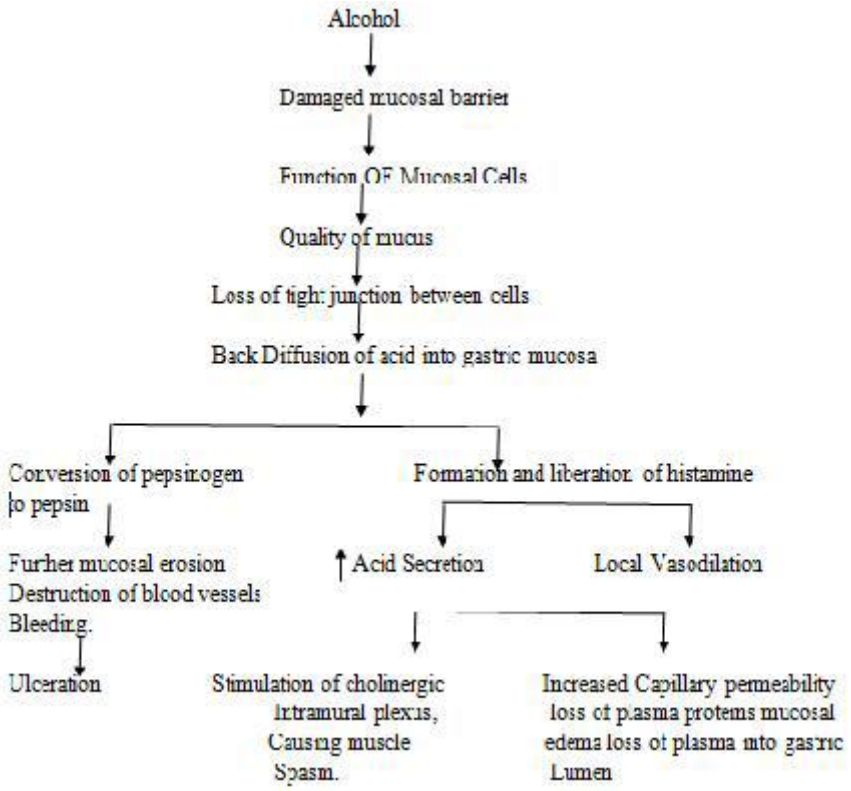


Fig. 1: Paradigm of Pathology of Gastritis.

The excessive consumption of drinks containing ethyl alcohol is generally believed to induce acute or chroni gastritis, the latter often being described as "atrophic"; moreover, many clinicians attribute the dyspeptic manifestations of alcoholism to such inflammation. Theoretically, gastritis is a reasonable expectation, and there is reliable evidence that it does occur in alcoholic subjects. However, in a recent personal histological study of gastric mucosa from such subjects inflammatory lesions were notably slight or entirely absent, even in some inveterate spirit-drinkers.[26] The effects of ethanol on gastric emptying are probably dependent on the beverage: a low alcohol dose (wine and beer) seems to induce gastric motility whereas higher alcohol concentrations delay gastric emptying.[27] The latter may produce a sensation of epigastric fullness or even nausea.

Alcohol Metabolism: about 20% of Ethanol ingested is absorbed into the stomach wall by passive diffusion and rest 80% is absorbed through duodenum and intestine wall.[28,29] liver contains dehydrogenase (ADH), aldehyde dehydrogenase (ALDH), cytochrome P450 2E1 (CYP2E1), and othr catalase which helps in the oxidation of ingested alcohol in to their metabolite in normal adults.[30] but a second pathway takes place when the person is a chronic alcoholic that is smoth endoplasmic reticulum contain the microsomal ethanol‐oxidizing system (MEOS) in which convert the alcohol to acetaldehyde and helps to get rid of the toxic compounds in the body via CYP2E1.[31] This reaction produces NADP and water with help of oxygen and reduced nicotinamide adenine dinucleotide phosphate (NADPH).[32] In some cases, a small amount (2%) of ethanol are oxidizes into acetaldehyde by the catalase located in the peroxisomes in the presence of a hydrogen peroxide (H2O2)‐generating system [Figure 2].[33] and as a cofactor nicotinamide adenine dinucleotide (NAD) not require for catalase.[34]

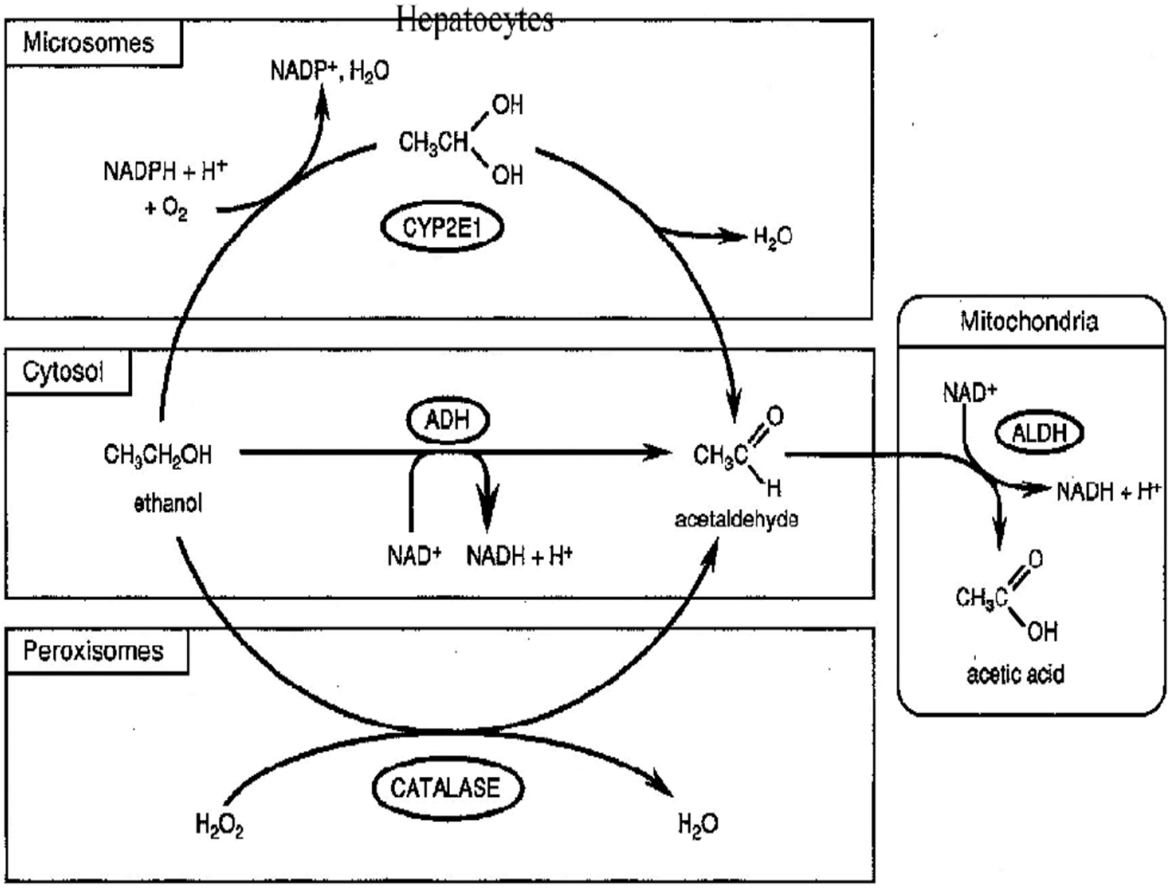


Figure. 2: Ethanol metabolism ADH = Alcohol dehydrogenase, ALDH = Aldehyde hydrogenase. Adapted from Liu, 2014.

Acetaldehyde can form adducts by binding to the different proteins such as enzymes, microsomal proteins, and microtubules and hence effect the functions of proteins or enzymes like protein adducts in the hepatocytes leads to hepatomegaly.[36] Acetaldehyde can also bind with brain signaling chemicals and leads alcohol dependence e.g. salsolinol is formed from dopamine and it also bind with the nucleic acid DNA and turns into carcinogenic eg. 1, N2‑propanodeoxyguanosine.[36]

alteration in the hepatic glycoprotein secreation caused due to alcohol is also mediated by this metabolite,acetaldehyde. in a preclinical animal study hepatic glycoprotein secreation impairment due to alcoholism is found to be mediated by acetaldehyde in rats with induced hepatic inflammation.[38] Acetaldehyde leads to injury over electron transport and oxidative phosphorylation by altering the electron transport chain (ETC) function and thereby injuring the production of reaction oxygen species (ROS) in which ETC complexes subunits are oxidises and[39,40] and which in turn decreasing the ATP levels.

Alcohol contribute to the accumulation of fat on the liver because it is rich in calories[41] and devoid of nutrients and it also decreases the food absorption and nutrients from the intestine. stomach and intestine tract can rapidly absorb into the sysytemic circulation because of the lipophilic nature of alcohol.[42] astric mucosa had vascular endothelium injury and becomes edematous and congestive by the high concentration of alcohol. alcohol exposure turns the principal and parietal cells to swollen and diminished,as they are rich in mitochondria and turns into mtDNA and is the target of ethanol associated intracellular oxidative stress[43,44] and hence causes to metabolic acidosis, cellular edema, intracellular calcium overload, and moreover gastric mucosa cell damages.[45] Also mucosal injury can lead by protein denaturation or enzyme inactivation and receptor damage or cell membrane modification caused due to oxidation of protein sulfhydryl groups present in gastric mucosa which are the target of ROS(reactive oxygen species).[46]

In addition to its effects on the tubular gastrointestinal tract, alcohol affects the physiologic motor action of the esophagus, stomach and intestine. In low concentrations it has a direct stimulating effect on gastric acid secretion, although contrary to conventional wisdom, it is not considered to have a pathogenetic influence on the development of peptic disease. Alcohol has a bactericidal effect on Salmonella, shigella, and H. pylori. It also has a tumorigenic role.

Gastritis is the inflammation in the stomach lining caused due to the alcohol; spicy and acidic food intake long-term consumption of nonsteroidal anti-inflammatory drugs (NSAID) and with severe bacterial infections, e.g. *helicobacter pylori* (*H. pylori*) and other chronic bile reflux, stress, certain autoimmune disorders, or the toxic substances such as carbon disulphide, asbestos, and iodoacetate.

Chronic gastritis is related to ulcer and gastric cancer.[48] Gastric mucosal erosion (disruption in mucosal defenses) is termed erosive gastritis. Gastritis reduces the gastric acid secretion. Gastritis in the corpus (corpus predominant and type A gastritis) and the antrum (antrum predominant or type B gastritis) behave differently, i.e. type A gastritis is more related to gastric carcinoma and type B gastritis is more related to ulcer disease. Pangastritis results from antrum-predominant chronic gastritis and it may also play a[49] pivotal role in alcohol-induced gastritis. The metabolic product of alcohol-aldehyde is a well-known carcinogen and plays a major role in alcohol-induced gastritis.[50,51]

Symptoms of gastritis

The symptoms of gastritis include indigestion, abdominal bloating, nausea, vomiting, pernicious anemia, burning, hiccups, loss of appetite, and black and starry stools.

Anti-inflammatory drugs, proton pump inhibitors (PPI), antacids, and antibiotics are used to treat gastritis.

Gastritis management is done by avoiding acidic foods, antacids supplements, and elimination of irritating foods like lactose, gluten, etc.[51-54]

Medications for gastritis

In case of *H. pylori* infection, "triple therapy," including PPI to reduce acid production and two antibiotics is given, otherwise Bismuth salicylate (Pepto-Bismol) is replaced by the second antibiotic. PPI decrease gastric acid production. PPI includes the following drugs: Esomeprazole (Nexium), lansoprazole (Prevacid), omeprazole (Prilosec), pantoprazole (Protonix), and rabeprazole (Aciphex).

Antacids may affect the absorption of the mediations; thereby decreasing the medicine's effectiveness. Antacids include aluminum hydroxide (Amphojel, AlternaGEL) magnesium hydroxide (Phillips'Milk of Magnesia), aluminum hydroxide and magnesium hydroxide

(Maalox, Mylanta) calcium carbonate (Rolaids, Titralac, and Tums), and sodium bicarbonate (Alka‑Seltzer). H2 blockers reduce gastric acid secretion. They include cimetidine (Tagamet), ranitidine (Zantac), nizatidine (Axid), and famotidine (Pepcid).[55-58]

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