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PEPTIC ULCER DISEASE: INSIGHTS FROM THE UNANI SYSTEM OF MEDICINE

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ABSTRACT

Peptic Ulcer Disease (PUD) is a chronic gastrointestinal condition characterized by ulceration in the mucosal lining of the stomach or duodenum, primarily due to an imbalance between gastric acid secretion and mucosal defense mechanisms. In the Unani system of medicine, PUD correlates with conditions like *Qarha-e-Hazmiyah*, which are attributed to imbalances in the humoral temperament, especially dominance of Safra (yellow bile) or Sauda (black bile). The disease is closely linked to *Su-e-Mizaj Meda* (derangement of gastric temperament), often aggravated by improper diet, stress, and lifestyle. The Unani approach emphasizes *Usool-e-Ilaj* (principles of treatment) such as *Izala-e-Sabab* (removal of cause), *Tadeel-e-Mizaj* (restoration of temperament), and the use of *Muqawwi-e-Meda* (stomach tonics) etc. Herbs like *Gul-e-Surkh*, Sandal, and Asl-us-soos are traditionally employed for their cooling, demulcent, and anti-inflammatory properties.

This paper explores the pathophysiology, diagnosis, and

management of Qaraha-e-Hazmiyah (PUD) within the Unani framework, highlighting its holistic and preventive approach rooted in centuries of traditional practice.

KEYWORDS: Peptic ulcer, Qarha-e-Hazmiyah, Temperament, Gastric mucosa.

INTRODUCTION

A Peptic ulcer is defined as disruption of the mucosal integrity of the stomach or duodenum leading to a local defect or excavation due to active inflammation. Ulcers occur within the stomach or duodenum are often chronic in nature.[1] Ulcers in the stomach or duodenum may be acute or chronic, both penetrate the muscularis mucosa but the acute ulcer shows no evidence of fibrosis. Erosions do not penetrate the muscularis mucosa.[2]

Gastric and duodenal ulcers coexist in 10% of patients and more than one peptic ulcer is found in 10-15% of patients. Peptic ulcer is a common disease in South India where the staple diet contains rice and less common in states where the staple diet is wheat. Duodenal ulcer is more common than GU; about 75 to 80% of the peptic ulcers are found in the duodenum.[3]

The annual global incidence rates of Peptic Ulcer were 0.10-0.19% for physician-diagnosed peptic ulcer disease and 0.03-0.17% when based on hospitalization data. The 1-year prevalence based on physician diagnosis was 0.12-1.50% and that based on hospitalization data was 0.10-0.19%.[4]

UNANI CONCEPT

The *Nazariya-e- Akhlat* (Humoral Theory), attributed to **Buqrat (Hippocrates- 460-370 BC),** serves as the foundational concept in Unani medicine. This theory posits that human health is governed by the balance of four essential bodily humors: *Dam* (blood), *Balgham* (phlegm), Safra (yellow bile), and *Sauda* (black bile). The disturbance in the equilibrium of these humors results in the development of disease. According to Unani Medicine, three primary factors contribute to the pathogenesis of diseases; altered temperament (*Su-e-Mizaj*), altered structural composition of the body (*Su*-e-*Tarkeeb*), and discontinuity of tissues and organs (Tfarruq-e-Ittisal).[5-9]

Classical Unani texts distinguish between various gastrointestinal ulcers, specifically *Qarha-e-ama* (intestinal ulcer) and *Qarha-e-Meda* (gastric or stomach ulcer), each described with unique etiologies and clinical features. The term "Qarha" in Unani terminology refers to a

purulent lesion or ulcerative wound affecting any muscle or organ, implying a breach in tissue integrity. Additionally, the term "Jarahat." is used more broadly to denote any wound or traumatic injury involving muscular structures."

In Unani medicine, an ulcer is defined as a pathological discontinuity affecting an organ, particularly those with muscular structure, and is often associated with purulent discharge.[10] The distinguished Unani scholar **Allama Qarshi (1210–1288 AD)** referred to *Qarha-e-Hazmiyah* (peptic ulcer) as a breach in the lining of the stomach, including the duodenum. This condition is believed to result from various causative factors, such as the accumulation of abnormal secretions from other organs into the stomach, the mixing of bile with phlegm, or the presence of corrosive and irritant phlegmatic substances.[11]

Ibne Sina (Avicenna-980-1037 AD) further explained that *Qarha-e-Meda* (Gastric Ulcer) is caused by the corrosive nature of certain humours *(Khilte Haad)*, which damage the gastric mucosa and compromise its continuity.[10]

HISTORICAL BACKGROUND

Peptic ulcer disease has been recognized since ancient times, though the precise origins of its medical documentation remain uncertain. Early references suggest that it may have been described by the ancient Egyptians or by Hippocrates (460–370 BC), with further mention attributed to Diocles of Carystus in the 4th century BC.[12,13]

Celsus provided one of the earliest known descriptions of gastric ulceration around 30 AD in his work De Medicina. He discussed dietary rules for maintaining health and noted that ulceration could affect the stomach if certain guidelines were not followed.[14]

Throughout history, references to symptoms such as indigestion and heartburn have been noted. However, it was not until the 16th century that autopsy confirmed the pathological basis of peptic ulcers. In 1586, Donatus of Mantua documented one of the first verified cases of pyloric peptic ulcer. Later, in 1679, Bauhin suggested that inflammation of the stomach could lead to ulceration and eventual rupture. The earliest documented case of gastric hemorrhage dates back to 1704.[12,13]

A more systematic classification of stomach diseases appeared in 1793 through the work of Matthew Baillie, who detailed inflammation, ulcers, perforation, pyloric stenosis, and ulcerated cancer. In 1817, Crampton described patients with perforated Gastric ulcers in

Dublin, while around the same time, Travers in London documented cases of bleeding, stenosis, and penetrating Gastric ulcers.[15,16]

Although ulcer disease has been recognized in medical writings for centuries, it only emerged as a distinct clinical diagnosis during the late 19th century. This increased recognition may reflect either a true rise in disease prevalence or an evolution in diagnostic interpretation, whereby symptoms previously attributed to dyspepsia or gastritis were reclassified as manifestations of peptic ulcer disease. Moreover, historical variations in the pathogenicity and prevalence of *Helicobacter pylori* likely contributed to shifting disease patterns. Certain populations, such as ethnic Malays, demonstrated notably low incidences of peptic ulcer and gastric carcinoma, correlating with a low prevalence of *H. pylori* infection. It has been hypothesized that *H. pylori* was either absent or non-virulent in Western populations until it gained virulence and spread during the 18th and 19th centuries. Remarkably, the earliest known case of peptic ulcer disease was identified in a mummified body from the Western Han Dynasty (167 BCE) in China, where autopsy findings revealed a perforated prepyloric ulcer complicated by acute diffuse peritonitis and disseminated intravascular coagulation.[12,13,17-19]

In the 1st century AD, ***Jalinoos*** (Galen) provided descriptions of *Qarah-e-Meda*, outlining its causes, clinical features, and anatomical location. Subsequently, **Rabban Tabri** (770 AD) expanded on this knowledge by detailing the causation, symptomatology, and treatment of Qarah-e-Meda. This foundational understanding was further elaborated by notable Unani scholars including **Razi** (850 AD), **Ibn Sina** (980 AD), **Majoosi** (994 AD), **Zahrawi** (1013 AD), and **Azam Khan** (1813 AD), who contributed significantly to the classical literature on gastrointestinal ulceration.[6,8,10,20,21]

ETIOPATHOGENESIS

1. Hereditary: Strong family history is observed with gastric ulcers, while duodenal ulcers show a less pronounced hereditary tendency. Genetic factors influencing acid secretion, mucosal defense, and inflammatory response may contribute to this predisposition.

2. Acid–pepsin versus mucosal resistance: The cause of peptic ulceration lies in the digestion of the mucosa by acid and pepsin of gastric juice. Normally, the stomach is capable of resisting this autodigestion. Thus, the concept of ulcer formation depends on the balance

between acid plus pepsin and mucosal resistance. Any factor that disturbs this equilibrium in favor of acid and pepsin can lead to ulcer formation.

3. Gastric hypersecretion: Ulcers occur only in the presence of acid and pepsin. Severe ulceration is seen in Zollinger–Ellison syndrome, which is characterized by markedly increased acid secretion. Acid secretion plays a more significant role in the etiology of duodenal ulcer than in gastric ulcer.

4. Mucosal resistance: Several mechanisms protect the gastric mucosa from acid injury. Surface epithelial cells secrete bicarbonate under the influence of mucosal prostaglandins, which neutralizes acid at the epithelial surface. These cells also secrete mucus that impedes the diffusion of ions and molecules such as pepsin. Prostaglandins play a central role in maintaining mucosal resistance by regulating the release of bicarbonate and mucus, inhibiting parietal cell secretion, and maintaining mucosal blood flow. This explains the ulcerogenic potential of NSAIDs.

5. Factors reducing mucosal resistance: Certain drugs, particularly those used in rheumatoid arthritis, reduce mucosal resistance. Aspirin is an important etiological factor in gastric ulcer; it damages cell membranes and tight junctions and inhibits prostaglandin synthesis, thereby reducing bicarbonate secretion and impairing mucosal defence.

6. Helicobacter pylori infection: The most common cause of peptic ulcer disease. The bacterium colonizes gastric mucosa, producing urease, cytotoxins, and proteases that damage epithelial cells and impair mucosal defense, leading to chronic gastritis and ulceration.

7. Drugs: NSAIDs and corticosteroids reduce mucosal prostaglandins and bicarbonate secretion, weakening mucosal protection and predisposing to ulcers.

8. Smoking: Nicotine increases gastric acid secretion, reduces bicarbonate production, and impairs mucosal blood flow, delaying ulcer healing.

9. Stress: Severe physiological stress (burns, trauma, sepsis) may cause acute stress ulcers due to mucosal ischemia and acid back-diffusion.

10. Dietary factors: Spicy foods, alcohol, and irregular meals may aggravate symptoms but are not direct causes. However, alcohol can injure mucosa and delay healing.[22,23,24]

Etiopathological Basis of Peptic Ulcer Disease According to Unani Medicine

In Unani medicine, disease is perceived as a systemic imbalance rather than a localized pathology. Health is maintained through a state of harmonious equilibrium among the four humors; Dam (blood), Balgham (phlegm), Safra (yellow bile), and Sauda (black bile), each possessing a specific temperament (Mizaj) and quality (Kaifiyat). The proper proportion and purity of these humors ensure the preservation of normal physiological functions, structural integrity, and vitality of the body. When this equilibrium is disturbed due to intrinsic or extrinsic factors, the homeostasis of the humoral system is disrupted, leading to a pathological condition known as Su-e-Mizaj (derangement of temperament). This derangement may manifest at the level of a specific organ or throughout the body, altering its structure and function. The Unani scholars therefore emphasized that the root of every disease lies in the qualitative or quantitative alteration of humors, which disturbs the temperament and functional harmony of the body.[25]

Classification of Disease According to Unani Concept

The etiopathogenesis of all diseases, according to Unani philosophy, is broadly classified into three principal types:

1. **Su-e-Mizaj (Sada or Maddi):** Refers to an alteration in the normal temperament of an organ or the whole body. *Su-e-Mizaj Sada* occurs without the involvement of morbid matter (*Madda*). *Su-e-Mizaj Maddi* is accompanied by the accumulation of abnormal humors that obstruct or impair physiological processes.
2. **Su-e-Tarkeeb:** Denotes a disturbance or deformation in the structural composition of an organ or tissue, often following chronic inflammation, degeneration, or sustained humoral imbalance.
3. **Tafarruq-e-Ittisal:** Represents the loss of continuity or integrity of tissues—resulting in ulceration, fissuring, or perforation of an organ's surface.[5-7,26]

Mechanism of Ulcer Formation (Qarah)

According to the above classification, ulcer formation (Qarah) primarily falls under Tafarruq-e-Ittisal, as it involves the disruption in the continuity of the mucosal or muscular layer of the stomach and intestines. However, this process is rarely isolated—it is typically preceded by Su-e-Mizaj (functional derangement) and Su-e-Tarkeeb (structural alteration). Hence, peptic ulcer disease is viewed as a sequential pathological process in which humoral imbalance first disturbs function, then structure, ultimately causing a breach in tissue integrity.[7,10]

This loss of continuity may arise due to external causes, such as trauma, ingestion of irritant substances, or heat exposure, and internal causes, which are more commonly implicated in gastric ulceration. The internal causes as described by Unani physicians include:

- Corrosive and caustic humors that chemically erode the mucosal lining.
- Moistening and relaxing humors (Ratab wa Mulaṭṭif Akhlat) that reduce tone and elasticity of the gastric wall.
- Drying and cleaving humors (Yabis wa Mushaqqiq Akhlat) that produce fissures and cracks within the mucosa.
- Gaseous distension caused by retained or penetrating gases that exert pressure on the stomach wall.
- Repletion of humors leading to congestion, inflammation, and subsequent ulceration.[27,28]

Role of Abnormal Akhlat and Temperamental Changes

Classical Unani physicians such as Ibn Sina, Razi, and Azam Khan have described that the accumulation of superfluous or morbid humors within the stomach alters its temperament and function. The predominance of Ghair Tabai Barid wa Ratab Mizaj (abnormally cold and moist temperament) particularly weakens the digestive power (Quwwat-e-Hazima), leading to delayed digestion, increased fluid retention, and impaired tissue resilience. This cold and moist state diminishes the stomach's innate resistance (Quwwat-e-Mudafiat), making it susceptible to ulceration. As the accumulation and stagnation of morbid Akhlat persist, they progressively Cause Su-e-Mizaj (functional derangement) by impairing digestive and secretory functions, Lead to Sue-e-Tarkeeb (structural alteration) due to prolonged inflammation and degenerative changes and Culminate in Tafarruq-e-Ittisal (loss of continuity) of the Ghisha-e-Mukhati wa Jirm-e-Meda wa Ama (mucous membrane and muscular wall of the stomach and intestine), resulting in ulcer formation.

Integrative Understanding

Thus, in the Unani framework, peptic ulcer disease (Qarah-e-Meda) is not a sudden or localized event but the end stage of a progressive pathological process arising from humoral disequilibrium. The sequential progression—from humoral imbalance → functional derangement (*Su-e-Mizaj*) → structural disorganization (*Su-e-Tarkeeb*) → breach of continuity (*Tafarruq-e-Ittisal*)—provides a holistic etiopathological explanation.

This classical concept aligns remarkably with modern medical understanding, which attributes ulcer formation to the breakdown of mucosal defenses, excessive acid secretion,

impaired repair mechanisms, and inflammatory injury. The Unani notion of weakened *Quwwat-e-Mudafiat* parallels the modern idea of compromised mucosal resistance, while *Barid wa Ratab Mizaj* can be correlated with reduced perfusion and impaired regenerative capacity of the gastric mucosa.[27,28]

CLINICAL FEATURES

Abdominal pain is common to many gastrointestinal disorders, including Duodenal ulcer (DU) and GU(GU), but has a poor predictive value for the presence of either DU or GU, up to 10% of patients with NSAID induced mucosal disease can present with a complication (bleeding, perforation, and obstruction) without antecedent symptoms. Despite this poor correlation, a careful history and physical examination are essential components of the approach to a patient suspected of having pep tic ulcers.

Epigastric pain described as a burning or gnawing discomfort can be present in both DU and GU. The discomfort is also described as an ill-defined, aching sensation or as hunger pain. The typical pain pattern in DU occurs 90 min to 3 h after a meal and is frequently relieved by antacids or food. Pain that awakes the patient from sleep (between midnight and 3 A.M.) is the most discriminating symptom, with two-thirds of DU patients describing this complaint. Unfortunately, this symptom is also present in one-third of patients with NUD (Non ulcer dyspepsia). The pain pattern in GU patients may be different from that in DU patients, where discomfort may actually be precipitated by food, Nausea and weight loss occur more commonly in GU patients. Endoscopy detects ulcers in <30% of patients who have dyspepsia.

Variation in the intensity or distribution of the abdominal pain, as well as the onset of associated symptoms such as nausea and/or vomiting, may be indicative of an ulcer complication. Dyspepsia that becomes stant, is no longer relieved by food or antacids, or radiates to the may indicate a penetrating ulcer (pancreas). Sudden onset of severe generalized abdominal pain may indicate perforation. Pain worsening with meals, nausea, and vomiting of undigested food suggest gastric outlet obstruction. Tarry stools or coffee-ground emesis indicate bleeding.[1,2]

PHYSICAL EXAMINATION

Epigastric tenderness is the most frequent finding in patients with GU or DU. Pain may be found to the right of the midline in 20% of patients. Unfortunately, the predictive value of this

finding is rather low. Physical examination is critically important for discovering evidence of ulcer complication. Tachycardia and orthostatic dehydration secondary to vomiting or active gastrointestinal blood loss. A severely tender, boardlike abdomen suggests a perforation. Presence of a succussion splash indicates retained fluid in the stomach, suggesting gastric outlet obstruction.

DIAGNOSIS

The clinician is often confronted with having to establish the presence of an ulcer. Documentation of an ulcer requires either a radiographic (barium study) or an endoscopic procedure. However, a large percentage of patients with symptoms suggestive of an ulcer have NUD, empirical therapy is appropriate for individuals who are otherwise healthy and <45, before embarking on a diagnostic evaluation. Barium studies of the proximal gastrointestinal tract are still commonly used as a first test for documenting an ulcer. The sensitivity of older single-contrast barium meals for detecting a Duodenal Ulcer is as high as 80%, with a double-contrast study providing detection rates as high as 90%. Sensitivity for detection is decreased in small ulcers (<0.5 cm). Benign GU also appears as a discrete crater with radiating mucosal in-folds originating from the ulcer margin. Ulcers >3 cm size of those associated with a mass are more often malignant Unfortunately, up to 8% of GU that appear to be benign by radiographic appearance are malignant by endoscopy or surgery. Radiographic studies that show a GU must be followed by endoscopy and biopsy Endoscopy provides the most sensitive and specific approach for examining the upper gastrointestinal tract. In addition to permitting direct visualization of the mucosa, endoscopy facilitates rule out malignancy (GU) or H. pylori. Endoscopy, and Biopsy examination particularly helpful in identifying lesions too small to deciation.[1,2,3]

There are four natural types of medah (stomach) accordingly, Haar, Barid, Yabis and Ratab. Diagnosis of disease is made by comparing the original and altered mizaj in Unani system. Original mizaj of medah is Garm-wa- Tar (hot and moist) as it is a muscular organ.[29]

UNANI MANAGEMENT

In the Unani system of medicine, the management of gastritis, gastric ulcers (GU), and related gastrointestinal disorders involves the use of drugs possessing stomachic (Muqawwī-e-Mi‘da), astringent (Qābid), desiccant (Mujaffif), styptic (Hābis-e-Dam), sedative (Musakkin), and coolant (Mubarrid) properties.[30] These agents help to strengthen the stomach, reduce inflammation, promote healing of ulcers, and restore the balance of gastric

temperament. A wide range of herbs and natural substances have been identified for their protective and healing effects on the gastric mucosa and peptic ulcers. Herbal drugs from the Unani pharmacopeia have demonstrated activities such as reducing gastric acidity, enhancing mucosal defense, and accelerating ulcer healing.[31]

Usoole Ilaj Wa Ilaj (Principles of Treatment)

The Unani system of medicine follows a holistic and well-structured approach in the management of diseases, emphasizing the correction of underlying imbalances rather than merely addressing symptoms. The guiding principles for the treatment of ulcers and gastrointestinal disorders are as follows:

1. Izāla-e-Sabab (Removal of Causative Factors): The primary step in treatment is to identify and remove the root cause of the disease. This may involve correcting dietary indiscretions, modifying lifestyle habits, or eliminating environmental factors that contribute to gastric irritation or ulcer formation.

2. Nuzj wa Istifrāgh (Concoction and Evacuation): This process includes Nuzj, the ripening or maturation of morbid matter to prepare it for expulsion, followed by Istifrāgh, the elimination of these humoral impurities through suitable routes such as emesis, purgation, or other regimental procedures. This helps restore humoral balance and supports mucosal healing.[35]

3. Use of Agglutinant (Mugharrirāt) and wound-healing (Mudammilāt): The use of irritant or corrosive drugs is strictly avoided to prevent aggravation of gastric or oesophageal ulcers. Instead, Agglutinant and wound-healing agents such as *Samagh-e-Kateerā* (Tragacanth gum) and *Samagh-e-'Arbī* (Gum arabic) are prescribed to protect and repair the ulcerated mucosa.

4. Tanqiya-e-Qurūh (Cleansing of Ulcer): To clean the ulcer and remove impurities, purifying agents (Munqqiyāt) with detergent (Jālī) and soothing (Muzliq) properties are administered. Preparations like *Ma'ul 'Asal* (honey water) and *Julāb* (rose water syrup) are often used to gently cleanse and soothe the affected mucosa.

5. Tajfīf wa Indimāl (Desiccation and Healing): Once the slough and exudates have been cleared, desiccant (Jāzib) and healing (Mudammil) drugs are employed to facilitate tissue repair, promote granulation, and accelerate the recovery of the ulcerated surface.[6,27,28]

IMPORTANT UNANI COMPOUND FORMULATIONS FOR PEPTIC ULCER

COMPOUND DRUGS	INGREDIENTS
Qurs-e- kahruba	Kahruba (Amber) – fossil resin Tukhm-e-Khurfa (Purslane seeds) – Portulaca oleracea Kafoor Hindi (Camphor) – Cinnamomum camphora Kateera (Tragacanth gum) – Astragalus gummifer Samagh Arabi (Gum Arabic) – Acacia arabica Kishneez Khushk (Coriander) – Coriandrum sativum Khashkhash Safed (White Poppy seeds) – Papaver somniferum Nashasta Gandum (Wheat starch) – Triticum aestivum Aab-e-Bartang (Psyllium mucilage) – Plantago ovata.[32]
Qurs-e- gulanr	Gulnar (Pomegranate flower) – Punica granatum Gile Armani (Armenian bole) – Bolus Armena Samagh Arabi (Gum Arabic) – Acacia arabica Gule Surkh (Red Rose petals) – Rosa damascena Aqaqia (Babool gum / Acacia) – Acacia arabica Kateera (Tragacanth gum) – Astragalus gummifer Aab-e-Gulnar (Pomegranate) – Punica granatum[32,33]
Ayarij Faiqra	Sibr (Aloe veara) – Aloe barbadensis Mastagi (Mastic gum) – Pistacia lentiscus Ood-e-Balsan (Balsam wood) – Commiphora opobalsamum Darchini (Cinnamon) – Cinnamomum zeylanicum Saleekha (Carthamus) – Carthamus tinctorius Asarun (Wild Spikenard) – Asarum europaeum Habbe Balsan (Balsam resin pellet) – Commiphora opobalsamum Sumbul-ut-Tib (Musk-root) – Nardostachys jatamansi[10]

SOME SINGLE DRUGS(MUFRAD)

S. N	Unani Name	Botanical Names	Af'al	Chemical constituents	studies
1	Kalonji	Nigella sativa	Muqawwi meda (strenghthener of stomach), Jali (detergent), Munafis-e-Balgam (expectorant).	hymoquinone, Thymohydroquinone, Dithymoquinone, Nigellone, α-Hederin, Fixed oils (linoleic & oleic acids), Alkaloids (Nigellidine, Nigellimine), Flavonoids	Gastroprotective effect against the Indomethacin induced ulcer[34]
2	Asal	Honey	Munaffis-e- Balgham (expectorant), Mulattif (demulscent), Dafe Taffun (antiseptic), Muqawwi-e-Medah (strenghthener of stomach)	Fructose, Glucose, Sucrose, Organic acids (gluconic acid), Enzymes (invertase, catalase, glucose oxidase), Polyphenols (quercetin, galangin, pinocembrin), Amino acids, Vitamins & Minerals	Anti-oxidant and anti-inflammatory effect in the treatment of chronic ulcer and preservation of mucosal glycoproteins[35]
3	Asl-us-soos	Glycyrrhiza glabra	Munaffis-e- Balgham (expectorant), Mulattif (demulscent), Dafe-e-Taffun (antiseptic),	Glycyrrhiza glabra Glycyrrhizin, Glycyrrhizic acid, Liquiritin, Isoliquiritin, Glabridin, Liquiritigenin,	Hydroalcoholic extract of *Glycyrrhiza glabra* demonstrated a

			Muqawwi-e-Medah (strenghthener of stomach)	Flavonoids, Saponins, Polysaccharides	significant anti-ulcerogenic effect, mediated through the enhancement of gastric mucosal defensive factors.[36]
4	Samagh-e-arabi	Acacia senegal	Mujaffif (Desiccant), Qabiz (astringent)	Arabinogalactan, Arabinose, Rhamnose, Uronic acids, Polysaccharides, Calcium/Magnesium salts, Glycoproteins	Reduction in pro-inflammatory cytokines in plasma and gastric tissue resulting in enhanced gastric protection and preserved mucosal integrity.[37]
5.	Sandal safed	Santalum album L.	Muqavvi-e-meda (Strengthens the stomach)	α-Santalol, β-Santalol, Santalene, Santalic acid, Sesquiterpenoids, Tannins	Increase in gastric protection resulting in a significant decrease in both number and severity of ulcers[38]
6	Abebartang	Platango ovata	Kasir-e-riyah (carminative), Dafe-e-Taffun (antiseptic)	Mucilage (arabinoxylans), Iridoid glycosides (Aucubin, Catalpol), Flavonoids, Tannins, Phenolic acids (caffeic, chlorogenic), Polysaccharides	Aqueous extract of *Plantago ovata* seeds exhibits both anti-ulcer and hepatoprotective effects[39]
7	Kateera	Astragaluus gummifer	Muhallil (anti-inflammatory), Kabīsuddām (hemostatic), mudammil-e-qurūh (wound healing agent)	Polysaccharides (Tragacanthin & Bassorin), Arabinose, Xylose, Galacturonic acid, Rhamnose, Trace minerals	Anti-inflammatory, immunological, antioxidant and prebiotic effects[40]

Dietary recommendation

1. A diet comprising light and easily assimilable liquids is recommended. This includes:

- Aab-e- Anar (pomegranate juice)
- Aab-e-Santra (orange juice)
- Egg albumin
- Aashe Jaw (barley porridge)
- Sagudana (sago)

2. Diets that alleviate constipation (*Qabziyat*) and reduce the excessive heat (Hiddat) of bile-such as decoctions prepared from the branches of *Aleeq* and *Sambhalu*-are advised for individuals with a weakened digestive system.
3. *Taqleel-e-Ghiza* (Reduction in food intake) *Saree'ul Hazm Ghizaen* (Consumption of easily digestible foods)
4. Following *Tanqiya* (evacuation of morbid matter), it is essential to administer buttermilk, along with juices of *Safarjal* (quince) and *Rumman* (pomegranate), among others.
5. For *Tabreed/Tadeel-e-Mizaj* (Cooling and tempering the temperament)[27,28]

CONCLUSION

Peptic Ulcer Disease, referred to as Qarha-e-Hazmiyah in Unani medicine, is primarily attributed to an imbalance in the body's humors and disturbance in gastric temperament. Unani scholars emphasize the role of excessive heat and dryness, along with the accumulation of morbid matter, in the pathogenesis of ulcers. The Unani system adopts a holistic approach, focusing not only on symptom management but also on addressing the root cause through established principles such as *Izala-e-Sabab* (removal of the cause), *Tadeel-e-Mizaj* (correction of temperament), and *Tanqiya* (elimination of harmful substances). Dietary regulation and lifestyle modifications play a significant role in both treatment and prevention. From an integrative perspective, Unani concepts of altered temperament and mucosal weakness correspond closely with modern ideas of acid imbalance, mucosal defence failure, and *H. pylori* infection. Thus, Unani medicine provides a holistic and evidence-aligned framework for the management and prevention of peptic ulcer disease, focusing on restoring humoral balance and maintaining overall health.

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