

Annadravashoola-Parinamashoola (Acid peptic disease)

Acid-Peptic Disease (APD)

(Encompassing gastric & duodenal ulcer, erosive gastritis)

This write-up integrates contemporary gastro-enterology with Āyurvedic wisdom (primarily *Amlapitta / Pariṇāma-sūla* concepts). Standard modern sources: Bailey & Love (27th ed.), Sleisenger & Fordtran (11th ed.), ACG & Maastricht-VI guidelines. Classical Āyurvedic texts: Mādhava Nidāna-51, Caraka Saṃhitā Cikitsā-15, Aṣṭāṅga Hṛdaya Cikitsā-8.

1. Disease Description

Acid-Peptic Disease refers to mucosal injury of the oesophagus, stomach or duodenum caused by the caustic action of gastric acid, pepsin and (occasionally) bile.

- Core presentations:** epigastric burning/gnawing pain, meal-related dyspepsia, occult or overt GI bleed.
- Pathological spectrum:**
 - Gastric ulcer (GU)
 - Duodenal ulcer (DU)
 - Erosive / hemorrhagic gastritis
 - Gastroduodenitis (bulbar & pyloric channel)
 - Stress-related mucosal disease (SRMD)
 - Zollinger-Ellison hyper-secretory disease (rare)

2. Types & Salient Features

Broad Type	Site	Pain Timing	Key Notes
Duodenal ulcer	First part duodenum	Classically 2-3 h post-meal, relieved by food/milk	4× commoner than GU, nocturnal pain
Gastric ulcer	Lesser curvature antrum/body	Immediately after meal; food aggravates	Wt-loss frequent, malignancy must be excluded
Stress-ulcer	Fundus & proximal duodenum	Critically ill pts, painless bleed	ICU setting, burns, head-injury
NSAID-induced	Anywhere	Often asymptomatic until bleed	Elderly, concomitant aspirin
Refractory/Z-E	Multiple post-bulbar ulcers	Severe diarrhoea, high fasting gastrin	Gastrinoma

3. Major Aetiological Factors

- Helicobacter pylori infection** - 70-90 % DU; 40-60 % GU
- NSAIDs / Aspirin / steroids** - prostaglandin inhibition
- Gastric acid hyper-secretion - Z-E syndrome, systemic mastocytosis
- Lifestyle: smoking, alcohol, spicy meals (risk modifiers)
- Severe physiological stress - burns, sepsis, head injury
- Others: Crohn's, CMV/HSV (immunodeficiency), irradiation, post-Billroth surgery.

4. Etiopathogenesis (Modern Perspective)

1. Aggressive factors

- Acid (HCl) – parietal cell hyperactivity
- Pepsin – proteolytic mucosal injury
- *H. pylori* – produces urease → ammonium hydroxide → ↑ acid, ↓ somatostatin, cytokine-mediated injury
- NSAIDs – ↓ COX-1-mediated protective PGE₂ → ↓ mucus, bicarbonate, mucosal blood flow

2. Defensive factors

- Mucus-bicarbonate layer & surfactant phospholipids
- Adequate mucosal perfusion & prostaglandins
- Epithelial renewal & tight-junctions

Disease occurs when aggressors overwhelm defenders → epithelial break → ulcer crater → bleed / perforation.

5. Differential Diagnosis of Chronic Epigastric Pain

Condition	Distinguishing Clues
Gastro-oesophageal reflux (GERD)	Retro-sternal heart-burn, regurgitation, worse lying flat
Functional/Non-ulcer dyspepsia	Normal endoscopy, pain unrelated to meals
Biliary colic	RUQ radiation to back, ultrasound gall-stones
Pancreatitis (chronic)	Epigastric boring pain, steatorrhoea, CT changes
Gastric carcinoma	Persistent vomiting, early satiety, weight loss, anaemia
Cardiac angina (microvascular)	Exertional, relieved by nitrates, normal endoscopy

6. Diagnostic Work-up

1. **Bed-side** – Full history, NSAID/H-pylori risk, stool colour, anaemia signs.
2. **Laboratory** – CBC (microcytic anaemia), LFT, serum gastrin if refractory.
3. **Non-invasive H. pylori tests** –
 - Urea breath test (UBT) – gold standard for cure confirmation
 - Stool antigen ELISA
 - Serology (IgG) – only for screening.
4. **Upper GI endoscopy** (OGD) – FIRST-LINE if alarm features (>60 y, bleed, dysphagia, weight-loss). Allows biopsy for histology ± urease test.
5. **Imaging** – Barium meal (if endoscopy not feasible), CT for complications.
6. **Complication screening** – Fecal occult blood, rapid haemoglobin drop, erect X-ray (perforation).

7. Prognosis

- Uncomplicated APD heals in 4–8 weeks with PPI + *H. pylori* eradication – very good outcome.
- Recurrence <10 % after successful eradication.
- Risks: GI bleed (15 %), perforation (2–5 %), gastric outlet obstruction (2 %).
- GU may harbour malignancy → biopsy essential.

8. Modern Medical Management

1. General Measures

- Stop NSAIDs/aspirin where possible.
- Smoking & alcohol cessation, small frequent meals.

2. Acid Suppression

- a. Proton-pump inhibitors (PPI) – Omeprazole 20 mg OD / Pantoprazole 40 mg OD × 4–8 wks (↑ dose if large ulcer).
- b. H₂-receptor blockers – Ranitidine 150 mg BD (if PPI contraindicated).
- c. Antacids – Al(OH)₃ + Mg(OH)₂ PRN.

3. H. pylori Eradication (Maastricht-VI)

- Concomitant therapy × 14 days: – PPI std dose BD + Clarithromycin 500 mg BD + Amoxicillin 1 g BD + Metronidazole 500 mg BD

4. Mucosal Protectants

- Sucralfate 1 g QID before meals
- Bismuth sub-citrate 120 mg QID
- Misoprostol 200 µg QID (NSAID prophylaxis).

5. Treatment of Complications

- Endoscopic adrenaline/clip for bleed, IV pantoprazole infusion.
- Surgical repair – Graham patch (perforation), Vagotomy + antrectomy (rare now).

6. Maintenance

- Low-dose PPI in high-risk NSAID users.
- Repeat endoscopy for GU after 8 wks to ensure healing & exclude carcinoma.

9. Āyurvedic Perspective & Management

9.1 Rogābhinava (Correlation)

Modern Entity	Probable Āyurvedic Counterpart	Rationale
Duodenal Ulcer	Pariṇāma-śūla (Vāta-pittaja)	Pain in mid-digestion, relief by food
Gastric Ulcer / Dyspepsia	Ūrdhvaga Amlapitta	Sour eructation, burning soon after meals
Erosive Gastritis	Pittaja Āmā Chardi	Haematemesis possible

Correlation aids principle selection and not a strict one-to-one mapping.

9.2 Nidāna (Causative Factors)

- Ati-uṣṇa, ati-amlalavaṇa, viruddhāhāra
- Adhyāśana, irregular meal timing, night vigil
- Alcohol, smoking, anger, stress → Pitta + Vāta prakopa.

9.3 Saṃprāpti (Pathogenesis)

Nidāna → Tikṣṇa Pācaka-pitta ↑ + Samāna-vāyu vitiation → Vidagdha-pitta corrodes gastric duodenal mucosa → Vāta spasm → Pariṇāma-śūla / Amlapitta symptoms. Chronic corrosion leads to ulcer crater (*vrana*) + bleeding (raktapitta upadrava).

9.4 Saṃprāpti-Vighaṭana (Therapeutic Break-Up)

1. Pittāśamana (śīta-tikta-madhura dravya)
2. Vāta-anulomana (snigdha basti, ghṛta)
3. Mṛdu virecana to expel Vidagdha-pitta
4. Ropaṇa (ulcer healing) with ghṛta-kalpa, yashtimadhu, śata-dhauta ghṛta
5. Agni-dīpana (non-tikṣṇa) & grahaṇī-sthira karaṇa

9.5 Doṣaja Cikitsā-Sūtra

1. “Tikta-ghṛta-snehapānaṁ pitta-parikalpita-pariṇāma-śūle”
2. “Sūtasekharābhyaṅgaḥ mṛdu-virecana-pūrvah”
3. “Kṣīra-basti vāta-saṁsrṣṭe vyathe”
4. “Śīta-madhura-dravyaiḥ stambhanaṁ rakta-darśane”

9.6 Cikitsā-Yojanā (Protocol)

Stage	Therapy	Classical Yoga (Dose)	Notes
Poorva-karma	Snehapāna	Tikta-ghṛta 40–60 ml OD × 3–5 d	Monitor for sātmyata
Śodhana	Mṛdu Virecana	Avipattikara cūrṇa 8–10 g HS	Especially in adhoga amlapitta
Basti	Kṣīra-basti	Milk 300 ml + Ghṛta 50 ml + Yashtimadhu 10 g × 5 alt. days	For Vāta-associated pain
Śamana	-		
	Sūtaśekhara Rasa 125 mg TID with honey-ghee	Pittaja burning, nausea	
	Kāmdudha Rasa (Moti yukta) 250 mg BID with cool milk	Ulcer bleed prophylaxis	
	Praval-Pañcāmṛta 500 mg BID	Neutralises acid, provides Ca ²⁺	
	Dhātrī-Lauha 500 mg BID	Anti-ulcer + iron	
	Yashtimadhu Ghṛta 10 ml HS	Mucosal repair	
	Saṁskṛta Nārikel Lavan 1–2 g BD	Duodenal ulcer, excellent pH buffer	
Rasāyana	Śatāvārī-Ghṛta 10 ml HS × 1 month	Prevent recurrence, nourish duodenal lining	

9.7 Pathya-Apathya

PATHYA (Wholesome)

- Warm cow-milk with ½ tsp ghee before sleep
- Old shāli rice, barley, bottle-gourd, pumpkin, cucumber
- Pomegranate, ripe banana, sweet grapes
- Coriander-fennel infusion, coconut water
- Small, regular meals; sit erect 30 min post-meal; early dinner
- Yoga: Vajrāsana after meals, Sheetalī & Bhrāmārī prāṇāyāma

APATHYA (To Avoid)

- Pickles, tomato ketchup, vinegar, chillies, deep-fried snacks
- Sour curd at night, cola or energy drinks, smoking, alcohol
- Long fasting, late-night study, emotional outbursts
- Analgesic abuse (NSAIDs) without gastro-protection

10. Integrated Algorithm (Clinic Quick Sheet)

1. Alarm symptoms? → Urgent endoscopy ± PPI infusion
2. H. pylori +ve → PPI-based quadruple therapy + start Āyurvedic Pittāśamana (Kāmdudha) day-4 onward
3. No ulcer on OGD but dyspepsia → treat as functional + Amlapitta regimen (Avipattikara, Shatadhauta Ghṛta)
4. At 4 weeks reassess:
 - If asymptomatic & GU → repeat OGD to confirm healing
 - Begin Rasāyana (Śatāvārī-ghṛta) 1–2 months
5. Lifestyle & pathya counselling each visit to prevent relapse.

11. Prognostic Summary

Scenario	Modern Outlook	Āyurvedic Yukti
Uncomplicated DU	Heals >90 % with PPI & HP eradication	Śighra-sādhyā under Pitta-śamana & pathya
GU with benign histology	Good, needs repeat scope	Sādhyā; gṛta-kalpa prevents scarring
Refractory / Z-E	Requires life-long PPI ± surgery	Kṛcchra-sādhyā; dūṣya balāvipatti
Bleed / perforation	Mortality 5-10 %	Agantu-vyapad; post-op rasāyana vital

Key Classical Quote

“Pāke tu pariṇāme ca śūlaṃ yad upajāyate, Dugdha-sevane śamayati tam *Pariṇāma-śūla*.” – A.H. Cikitsā-10/38

Selected References

1. Malfertheiner P. et al. Management of *Helicobacter pylori*: Maastricht VI/Florence Consensus Report. *Gut* 2022.
2. Bailey & Love's Short Practice of Surgery, 27th Edition, 2018.
3. Mādhava-kara. *Mādhava Nidāna* with Madhukośa, Ch. 51.
4. Cakrapāṇi: *Charaka Saṃhitā* – Cikitsā-sthāna 15.
5. American College of Gastroenterology. Clinical Guideline: Diagnosis & Management of PUD, 2021.