

3.1 Peptic ulcer diseases

Peptic ulcer disease is ulceration of the mucosa of the stomach or duodenum extending through the muscularis mucosae, occurring where acid and pepsin overwhelm mucosal defence. The balance is the pathophysiology and the organising image of the topic: on the aggressive side, gastric acid, pepsin, bile reflux and external injurious agents; on the defensive side, the mucus bicarbonate barrier, mucosal blood flow, prostaglandin driven mucus and bicarbonate secretion and rapid cell renewal. The two dominant causes work one on each side. *Helicobacter pylori*, a spiral bacterium colonising the gastric mucus layer, causes chronic gastritis and duodenal inflammation that ulcerates; it causes most duodenal ulcers and a majority of gastric ulcers. Non steroidal anti inflammatory drugs fall on the defensive side: by inhibiting cyclo oxygenase they deplete the mucosal prostaglandins that maintain mucus, bicarbonate and blood flow, producing ulcers that are often silent until they bleed, the standard drug induced disease of this unit. Diagnosis is by upper gastrointestinal endoscopy, which sees the ulcer, excludes malignancy in gastric ulcers by biopsy and allows *H. pylori* sampling, supported by the urea breath test and stool antigen test in patients managed without endoscopy; alarm features, weight loss, anaemia, dysphagia, vomiting or older age, mandate endoscopy rather than empirical treatment.

Pharmacotherapy follows the balance: suppress acid so the mucosa can heal, eradicate *H. pylori* where present so the ulcer does not recur, and remove or protect against NSAID injury. Proton pump inhibitors, which irreversibly block the parietal cell hydrogen potassium ATPase, the final common step of acid secretion, are the most effective suppressants and the healers of choice; histamine H₂ receptor antagonists are weaker and now secondary. Eradication therapy combines a proton pump inhibitor with antibacterial agents in the standard multi drug regimens, given as a course whose completion matters more than any single component. Mucosal protectives have defined places: sucralfate forms an adherent barrier over the ulcer base, and misoprostol, a prostaglandin analogue, replaces the defence NSAIDs remove and is the specific prophylactic answer where NSAID therapy must continue at high risk, limited by diarrhoea and by contraindication in pregnancy. For NSAID ulcers the first step is to stop or reduce the NSAID where possible, add a proton pump inhibitor for healing and continuing protection, and consider a cyclo oxygenase 2 selective agent where anti inflammatory therapy is unavoidable, weighing gastrointestinal against cardiovascular risk.

Peptic ulcer: the aggressive and defensive balance

AGGRESSIVE FACTORS	THE ULCER FORMS WHEN	DEFENSIVE FACTORS
Gastric acid Pepsin Helicobacter pylori NSAID injury Smoking and alcohol	Aggression rises, or defence falls, or both Healing needs acid suppressed and the cause removed	Mucus bicarbonate barrier Mucosal blood flow Prostaglandins Rapid cell renewal

PPIs suppress acid, eradication removes H. pylori, misoprostol replaces prostaglandins

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Figure: The peptic balance. An ulcer forms when aggression rises or defence falls, and every therapy in Section 3.1 acts on one side of this balance.

3.2 Reflux esophagitis

Reflux esophagitis is inflammation of the oesophageal mucosa caused by reflux of gastric contents, within the wider gastro oesophageal reflux disease, in which reflux causes troublesome symptoms or complications with or without visible injury. The oesophagus has little defence against acid and pepsin, so reflux that would be harmless lower down injures it. The mechanisms are failures of the antireflux barrier and of clearance: transient relaxations and incompetence of the lower oesophageal sphincter, hiatus hernia, impaired peristalsis that fails to clear refluxate, delayed gastric emptying, and raised intra abdominal pressure from obesity and pregnancy; fatty meals, caffeine, alcohol, smoking and sphincter relaxing medicines such as calcium channel blockers and nitrates contribute. Untreated injury produces erosive esophagitis and, with chronicity, peptic stricture, Barrett oesophagus, in which squamous epithelium is replaced by intestinal type columnar epithelium with malignant potential, and adenocarcinoma arising in that segment. Presentation is heartburn after meals and on lying or bending, acid regurgitation, and dysphagia where stricture narrows the lumen; extra oesophageal presentations include chronic cough and laryngeal symptoms, and chest pain that must first be distinguished from cardiac pain. Diagnosis is usually clinical and confirmed by response to acid suppression; endoscopy grades esophagitis, detects Barrett change and is required for alarm features, and pH monitoring settles doubtful cases.

Pharmacotherapy aims to make the refluxate harmless, since the barrier itself is corrected mechanically, by surgery or endoscopic procedures, rather than by drugs. Proton pump inhibitors are the principal class, healing esophagitis more reliably than H₂ receptor antagonists, which retain a place in milder disease and for nocturnal symptoms. Treatment is stepped characteristically: a healing course of full suppression, then step down to the lowest level maintaining control, maintenance therapy where relapse or complications demand it, and on demand therapy for intermittent non erosive symptoms. Lifestyle measures are prescribed with the same seriousness: weight reduction where overweight, smaller and earlier evening meals, head of bed elevation for nocturnal symptoms, and avoidance of individual triggers, smoking and late alcohol.

3.3 Inflammatory bowel diseases

The inflammatory bowel diseases are ulcerative colitis and Crohn disease, two chronic relapsing inflammatory diseases of the gut whose comparison is a fixed examination structure. Both arise from an inappropriate, sustained immune response to the intestinal microbiota in a genetically susceptible host, with environment shaping course: smoking worsens Crohn disease and paradoxically appears protective in ulcerative colitis. Neither is a simple infection and neither is cured by removing a single cause. Ulcerative

colitis is continuous inflammation of the colonic mucosa beginning at the rectum, limited to the colon and the mucosal layer, producing a friable, ulcerated surface that bleeds readily. Crohn disease is transmural inflammation that may affect any part of the gut in discontinuous skip lesions, most often terminal ileum and colon, and its depth produces its defining complications: strictures, fistulae, abscesses and perianal disease. Presentation divides accordingly: ulcerative colitis chiefly with bloody diarrhoea, urgency and tenesmus in flares, severe attacks risking toxic megacolon; Crohn disease with pain, less bloody diarrhoea, weight loss and obstructive or fistulating complications.

Pharmacotherapy pursues two goals in sequence, inducing remission in the flare and then maintaining it. Aminosalicylates, delivering 5-aminosalicylic acid to the colonic mucosa, are the foundation in ulcerative colitis for mild to moderate flares and maintenance, by mouth and rectally where disease is distal; their place in Crohn disease is limited. Corticosteroids induce remission in moderate to severe flares of both diseases and are deliberately not maintenance therapy, because dependence and systemic harm forbid it. Immunomodulators, the thiopurines and methotrexate, maintain remission and spare steroids, acting slowly and requiring blood monitoring for marrow and liver toxicity. Biologic therapies, anti-TNF agents and the newer biologics against integrins and interleukin pathways, together with oral Janus kinase inhibitors in ulcerative colitis, treat moderate to severe disease failing conventional therapy, under specialist care with infection screening before immunosuppression. Surgery remains part of management: curative of the colonic disease in ulcerative colitis at the price of colectomy, and in Crohn disease reserved and bowel preserving, since recurrence after resection is the rule.

Feature	Ulcerative colitis	Crohn disease
Distribution	Colon only, continuous from the rectum	Any part of the gut, discontinuous skip lesions
Depth	Mucosal only	Transmural, full thickness
Hallmark complications	Bleeding, toxic megacolon, colorectal cancer risk	Strictures, fistulae, abscesses, perianal disease
Dominant presentation	Bloody diarrhoea with urgency	Pain, diarrhoea, weight loss, obstructive features
Surgery	Colectomy removes the colonic disease	Reserved, bowel preserving, recurrence common

3.4 Jaundice and hepatitis

Jaundice is the yellow discolouration of skin, sclerae and mucous membranes by accumulated bilirubin, a sign and not a disease, taught by its mechanism in three families. Bilirubin is produced from haem breakdown, carried to the liver bound to albumin, conjugated in hepatocytes and excreted in bile. Pre-hepatic jaundice follows overproduction, chiefly haemolysis, with a normal liver. Hepatocellular jaundice follows liver cell injury, in hepatitis and cirrhosis, where uptake, conjugation and excretion fail together. Post-hepatic or obstructive jaundice follows blocked bile flow from gallstones, strictures or tumours, with dark urine, pale stools and itch from retained bile salts. The clinical approach reads that pattern with liver enzyme patterns and imaging, ultrasound first for obstruction, to place the patient in a family and then a cause. Hepatitis is inflammation of the liver, and its causes define its pharmacotherapy. The hepatotropic viruses A to E are the classic family: A and E spread faeco-orally and cause acute self-limiting disease, E dangerous in pregnancy; B and C spread by blood, sexual contact and vertically, and may become chronic and progress to cirrhosis and hepatocellular carcinoma; D occurs only with B. Beyond viruses, hepatitis arises from alcohol, metabolic fatty liver disease, autoimmune hepatitis, inherited copper and iron storage disorders, and drugs, the subject of Section 4.3.

Pharmacotherapy of hepatitis is cause specific, and general measures come first: avoidance of alcohol and of unnecessary hepatotoxic medicines, and no drug therapy for uncomplicated acute viral hepatitis, which resolves with supportive care. Hepatitis A and B are vaccine preventable, and vaccination of contacts and risk groups is pharmacotherapy in preventive form. Chronic hepatitis B is treated with antiviral therapy where viral activity and liver injury criteria are met: nucleoside and nucleotide analogues suppressing replication over long courses, and interferon based therapy in selected patients, aiming to prevent cirrhosis and cancer. Chronic hepatitis C has been transformed by direct acting antivirals, oral combinations inhibiting the viral protease, polymerase and replication complex proteins that cure the great majority of treated patients over short courses, a clear therapeutic advance in this syllabus. Autoimmune hepatitis is treated with corticosteroids and steroid sparing immunosuppression, typically azathioprine. Alcoholic hepatitis is managed by abstinence above all, nutrition, and corticosteroids in selected severe cases under specialist judgement.

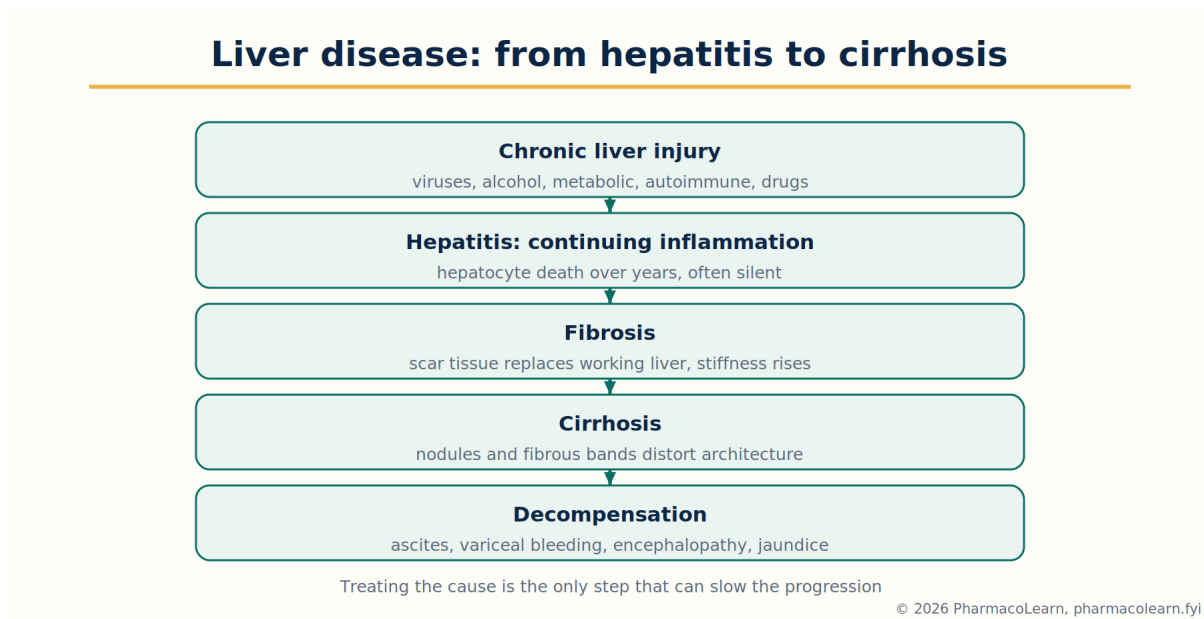


Figure: The road to cirrhosis. Hepatitis is the stage at which treating the cause can still change the destination.

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