Atlas of
Diagnostic Endoscopy
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SECOND EDITION

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JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD
Kolkata • St Louis (USA) • Panama City (Panama) • London (UK) • New Delhi
Ahmedabad • Bengaluru • Chennai • Hyderabad • Kochi • Lucknow • Mumbai • Nagpur
Published by
Jitendar P Vij
Jaypee Brothers Medical Publishers (P) Ltd

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Atlas of Diagnostic Endoscopy
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First Edition: 2004
Second Edition: 2011
ISBN 978-93-80704-50-0
Typeset at JPBMP typesetting unit
Printed at
Preface to the Second Edition

Each passing year has seen tremendous advances in the field of both diagnostic and therapeutic endoscopy. While preparing the current edition of the atlas, I also felt tempted to add a few chapters on recent advances like fluorescent endoscopy, magnification endoscopy, etc. However, on the second thought, I decided to restrict myself to the basic endoscopy since my target readers, as I mentioned in the first edition of the atlas, are the ‘young doctors who wish to get initiated and practice endoscopy’. The aim of this book is to provide them a strong foundation by familiarizing them with the basic concepts of endoscopy and aiding in correct interpretation of the pathology. Not withstanding the number of similar atlases available online, it is always quick and easy to refer to a printed copy that is lying in the endoscopist’s consultation chamber. Needless to say, printed images provide a longer lasting impression as compared to those seen on the computer screen. Effort has been made to replace some poor quality and repetitive images of the previous edition with new ones giving a fresh look to the current edition of the atlas. I sincerely hope that the atlas, in its current form, will find wide acceptance amongst the endoscopy practitioners.

Mohammad Ibrarullah
Some years ago, when I started my endoscopy practice, I was fortunate enough to have the backup of some experienced colleagues and a well-equipped institutional library having a few elegant books on the subject. But not all young doctors who wish to get initiated and practice endoscopy are as fortunate. ‘Can you suggest me some good atlas on endoscopy’? Is the usual request from most of them. I must confess that, despite my several years of practice I have not been able to gather the courage to possess a personal copy of even a single atlas. Almost all the available atlases are, by Western authors, expensive and largely inaccessible. In the absence of proper guidance, either from a senior colleague or from an atlas, the young endoscopist largely depends on his ‘clinical sense’ to interpret the endoscopic findings. Needless to say this results in serious errors and blunders. It is appalling to find esophageal varices being misdiagnosed as tumors or an advanced gastric carcinoma as erosive gastritis and so on. Hence, a great need was felt to compile one such atlas that is within the reach of the endoscopist, in this country and abroad, without of course compromising the contents and quality.

Mohammad Ibrarullah
Those who provided professional, academic and technical support in compiling the atlas are, Prof B Krishna Rau, Chennai; Prof G Subramanayam, Tirupati; Prof SR Naik, Lucknow; Dr D Srinivasa, Bengaluru; Dr Gajanan Wagholikar, Pune; Dr Anuj Sarkari, Gorakhpur; Dr Amarendra Mishra, Bhubaneswar; Dr Anwar Basha, Tirupati; Dr T Shyamsundar, Nellore; Dr B Visweswara Rao, Srikakulam; Dr D Vijay Nagaraj, Cudappa; Dr D Gopikrishna Reddy, Tirupati; Dr M Srinivas, Rajmundry; TL Varalakshmi, Tirupati; V Dhanalakshmi, Tirupati; Dr JM Rao, Bhubaneswar; Dr Neeraj K Mishra, Bhubaneswar; Dr Ambika P Das, Bhubaneswar; Dr Tapas Mishra, Bhubaneswar; Dr Sarat C Panigrahi, Bhubaneswar; Dr Devanand Mohapatra, Bhubaneswar; Dr S Shanmughanathan, Chennai; Gopala Bisoi, Bhubaneswar; Malaya Mukhi, Bhubaneswar.

The author wishes to thank Dr Sidhant Kar, Managing Director, Kar Clinic and Hospital, Bhubaneswar for his unconditional support in providing the modern equipment and the set-up to carryout the present work.
Chapter 1

TECHNIQUES OF UGI ENDOSCOPY AND NORMAL ANATOMY
PREPARATION FOR ENDOSCOPY

Informed consent and counseling: The patient should be clearly explained about the procedure and the likely discomfort he may experience. He should be convinced that his co-operation will make the procedure easier and quicker.

Overnight fasting: Routine endoscopy is usually performed in the morning hours after overnight fasting. Coating agents like antacids or colored medications should be clearly withheld. In case of obstructed stomach prior nasogastric intubation and lavage should be performed to clear the gastric residue.

Sedation and anesthesia: For routine UGI endoscopy we use only topical pharyngeal anesthetics like lignocaine viscus or spray. Sedation, in the form of intravenous midazolam is occasionally used in children. For therapeutic endoscopy like foreign body removal, stent placement etc., it is our practice to use intravenous propofol anesthesia with or without endotracheal intubation.

Endotracheal intubation and monitoring: Endoscopy in a comatosed or irritable patient is fraught with the risk of aspiration, hypoxia and 'bite' damage to the endoscope. It is our practice to use prior endotracheal intubation and also monitor the vital parameters during the procedure.

Instrument check: It is a good practice to check the instrument like light source, suction channel, airflow and display panel for any malfunction.

Position of the patient: Diagnostic endoscopy is always performed in left lateral position. Occasionally in a patient with upper GI bleeding, it may be necessary to examine the patient in right lateral position. This is to displace the blood pooled in the fundus that may obscure the bleeding lesion.

Antibiotic prophylaxis: Antibiotic prophylaxis is not indicated for diagnostic endoscopy. Current recommendations by American Society for Gastrointestinal Endoscopy (ASGE) exclude even conditions like valvular heart disease, prosthetic valves, synthetic vascular graft and prosthetic joints from the ambit of antibiotic prophylaxis. The few indications for antibiotic prophylaxis are, therapeutic endoscopy for cirrhosis with acute variceal bleeding, cyst drainage and in patients with established GI tract infection who have the above listed cardiovascular status.

Gastrointestinal Endoscopy 2008;67:791
The mouth-guard is held between the teeth (Fig. 1.1). It is further supported by the index and middle finger of the endoscopy assistant. Alternatively, an elastic band attached to the mouth-guard can be used to keep it steady.

The tip of the endoscope is slightly bent to fit the contour of the tongue. It is gently advanced over the base of the tongue towards the pharynx (Figs 1.2 and 1.3).
The epiglottis (E) is seen as the pharynx is entered (Figs 1.4A and B).

As the scope passes below the epiglottis, the larynx and both pyriform fossae come into view. The scope is kept in the midline at the esophageal inlet (arrow in Figs 1.5 and 1.6) and the patient is asked to take swallows. No undue force should be applied at this stage. Entry into the esophagus should be a voluntary effort supplemented by a gentle push by the endoscopist.
While negotiating esophageal inlet, such an appearance indicates passage of endoscope into trachea (Figs 1.7A and B). The patient becomes restless and starts coughing violently. Withdraw the endoscope at once. Reassure the patient and retry entering the esophagus after a while.

Esophageal mucosa is essentially featureless. The tracheal impression can be seen in the proximal esophagus (Fig. 1.8). Aortic impression and pulsation can be observed in the mid-esophagus (Fig. 1.9).
Z line represents the junction of pale squamous epithelium of esophagus with the pink columnar epithelium of stomach (Figs 1.10A to F). The junction may not be quite apparent when it lies at the level of diaphragmatic indentation (arrow in Fig. 1.10 A). In most of the cases, however, the junction can be made out clearly.

After crossing the GE junction the tip of the endoscope is slightly angled up and to the right. As the stomach is inflated, a tunnel (Fig. 1.11) becomes apparent. The roof and the base of the tunnel represent the lesser and greater curvature respectively. The endoscope is maintained close to the lesser curvature and gradually pushed forward. The mucosal rugosity in the gastric body turns flat marking the beginning of antrum (Fig. 1.12).
After inspecting the antrum the endoscope is directed towards pylorus. It is a common practice to cross the pylorus, examine D1, D2 and then come back to antrum and complete examination of the remaining stomach. Crossing pylorus is usually a frustrating experience for the beginner. In our practice, we advise the trainee endoscopist to use intravenous Hyoscine Bromide (Buscopan) to knock down gastric peristalsis, keep the pylorus in the center of vision, wait for the ring to open and then attempt to negotiate it. However, after a few endoscopies (usually 8-10) it ceases to be an issue and the endoscopist can cross the pylorus without much difficulty.

All the four walls of D1 are better visualized when the tip of the endoscope is placed at the pyloric ring (transpyloric view). Normally the D1 mucosa is featureless (Fig. 1.14).
The tip of the endoscope is impacted at the apex of D1 and rotated up and right. This maneuver facilitates its entry into D2. As endoscope is withdrawn slightly, its tip slips further down in to D3. The ampulla of Vater can be seen on the medial wall of D2. This is the distal extent of examination for routine UGI endoscopy.

The endoscope is now gradually withdrawn, carefully examining all the four walls of D2. The junction of D1-D2 is better inspected at this stage as the tip of the endoscope has a tendency to slip down during forward examination (Fig. 1.18).

Fig. 1.16A and B: Second part of the duodenum (D2) is marked by the circular mucosal folds

Figs 1.17A to C: Ampulla of Vater (arrow) seen on the medial wall of D2

Fig. 1.18: Junction of D1 and D2
The endoscope is withdrawn into antrum for examination of the remaining part of stomach (Figs 1.19A and B). The tip of the endoscope is flexed up bringing into view the incisura angularis (Fig. 1.19C). In this position, the endoscope is gradually withdrawn maintaining constant insufflation and slight rotation to the left. By this retroflexion or ‘J’ maneuver the entire lesser curvature can be inspected as the fundus is approached (Figs 1.20A to C). Fluid tends to accumulate in the fundus as this is the most dependant part of the stomach during endoscopy. This ‘fundic pool’ needs to be sucked out to have a clear view of the mucosa. The GE junction can be inspected from a close proximity by withdrawing and rotating the endoscope further (Figs 1.21A and B). Normally, the GE junction should appear snug around the shaft of the endoscope. This completes the examination of the upper GI tract. The tip of the endoscope is rotated to normal position, air in the stomach sucked out and the instrument withdrawn.
Chapter 3

HIATUS HERNIA AND GASTROESOPHAGEAL REFLUX DISEASE (GERD)
SLIDING HIATUS HERNIA—ENDOSCOPIC DIAGNOSIS

- Distance between the squamo-columnar junction and the diaphragmatic indentation (A and B respectively in Figs 3.1A and B) is > 2 cm. Normally, it is < 0.5 cm.
- On retroflexion, the diaphragmatic indentation (black arrows in Fig. 3.2) is not snug around the endoscope. The 'bell' like appearance represents the hiatal sac. Gastric mucosa appears drawn into the hiatal sac.
Figs 3.3A to D: Sliding hiatus hernia—view on retroflexion. (A) The diaphragmatic indentation (black arrows) is not snug around endoscope. The gastric mucosa has been pulled into the hiatal sac. (B, C) The squamo-columnar junction (white arrows) is above the diaphragmatic indentation. (D) Linear erosions in the esophageal mucosa stopping at squamo-columnar junction.

Fig. 3.4: Lax lower esophageal sphincter (LES)—view on retroflexion. Note the squamo-columnar junction (arrows) is at the level of diaphragmatic indentation. This feature differentiates it from sliding hiatus hernia despite the similarity in appearance between the two.
Figs 3.5A and B: Lax LES—view on retroflexion. Note the squamocolumnar junction (arrows) is almost coinciding with diaphragmatic indentation

Figs 3.6A to D: Lax LES with sliding hiatus hernia and associated dysmotility. (A) Food debris at the distal esophagus. The LES is open. (B-D) View on retroflexion
Hiatus Hernia and Gastroesophageal Reflux Disease (GERD)

Fig. 3.7: Gastric mucosal prolapse through lax LES

Fig. 3.8: Sliding hiatus hernia and gastric mucosal prolapse

Fig. 3.9: Sliding hiatus hernia and gastric mucosal prolapse

Fig. 3.10: Gastric mucosal prolapse through lax LES

Figs 3.11A and B: Sliding hiatus hernia and gastric mucosal prolapse
ENDOSCOPIC GRADING OF GERD

Savary-Miller Grading

Grade I
Oval or linear red patch situated above 'Z' line, often along a dorsal fold, may be covered with whitish exudate. Occasionally many such lesions are present, but they are not confluent.

Grade II
The erosive and exudative mucosal lesions are confluent but not involving the entire circumference.

Grade III
Involvement of entire circumference but stricturing is absent.

Grade IV
Presence of stricture or longitudinal shortening and/or the development of columnar metaplasia.

Los Angeles Grading

Grade A
One or more mucosal break(s) no longer than 5 mm, that does not extend between the top of two mucosal folds.

Grade B
One or more mucosal break(s) >5 mm long not extending between the tops of two mucosal folds.

Grade C
One or more mucosal breaks between the top of two or more mucosal folds involving <75% of the circumference.

Grade D
One or more mucosal break(s) involving at least 75% of the esophageal circumference.

Handbook and Atlas of Endoscopy
Solothurn, Schweiz:Gasman, 1978

Gut 1999; 45:172
Figs 3.14A and B: Erosions in the distal esophagus

Figs 3.15A and B: Erosions and exudates at GE junction

Figs 3.16A and B: Linear erosions at GE junction
Fig. 3.17: Erosions extending up to mid-esophagus

Fig. 3.18: Erosion extending proximally

Figs 3.19A and B: Linear ulcer extending from GE junction to proximal esophagus

Figs 3.20A to C: (A, B) Linear erosions. (C) Sliding hiatus hernia in the same patient
Figs 3.21A to D: Erosions extending up to squamocolumnar junction

Figs 3.22A and B: Sliding hiatus hernia. An ulcer at 6 O’clock position proximal to the squamo-columnar junction

Figs 3.23A and B: (A) Extensive ulceration involving the distal esophageal mucosa. (B) Gastrojejunostomy stoma in the same patient showing small erosion (arrow)
Figs 3.24A to D: (A, B) Esophagitis with sliding hiatus hernia. (C, D) View of the hiatal sac on retroflexion.

Figs 3.25A to C: Extensive esophageal involvement in GERD.
Concomitant peptic ulcer is not unusual in patients with severe esophagitis. Hyperacidity is found in 28% of patients suffering from GERD.

Figs 3.29 A to F: (A, B) Bile reflux esophagitis. Mucosal changes at the lower end of esophagus in a patient who had undergone gastrojejunostomy about 10 years back. (C) Hiatus hernia in the same patient. He was treated with Roux-en-Y conversion and partial fundoplication. (D, E) Normal appearing esophageal mucosa, six months after the surgery. (F) On retroflexion the gastric mucosa is tightly gripping the endoscope because of the antireflux procedure.

Figs 3.30 A to F: (A to C) Extensive ulceration involving distal esophagus. The patient underwent Nissen’s fundoplication. (D, E) Endoscopy three months after surgery showing healed esophageal ulcers. (F) Retroflexed view of the cardia subsequent to fundoplication.
Fig. 3.31A to D: Peptic stricture. (A) Fibrotic stricture at squamocolumnar junction. (B, C) Sliding hiatus hernia visible through the stricture. (D) Barium contrast study, in the same patient, showing stricture (arrow) at the distal esophagus and proximal dilatation. Note the diaphragmatic indentation (broken arrow) and the intervening hiatal sac.

Fig. 3.32: Peptic stricture in distal esophagus
Figs 3.33A to D: (A, B) Peptic stricture (arrow) and sliding hiatus hernia. (C, D) Close-up view of the stricture (arrow) on retroflexion showing fibrosis and nodularity.

Figs 3.34A to C: (A) Peptic stricture involving distal esophagus. Note the diverticulum (arrow) proximal to the stricture. (B, C) Sliding hiatus hernia in the same patient.

Figs 3.35A and B: Peptic stricture and esophageal ulcers.
Barrett’s esophagus is a complication of long standing GERD. It is characterized by flame-shaped or finger-like extension of gastric columnar epithelium into the esophagus for variable distance. It is considered a premalignant condition but the incidence of malignancy is extremely low. Four quadrant biopsy, every 2 cm, is recommended to detect dysplasia. Patients with mild and moderate dysplasia are treated with conventional anti-reflux treatment and kept under surveillance. High grade dysplasia is treated by surgical excision. Endoscopic mucosal resection (EMR) or mucosal ablation by—laser, argon plasma coagulation, multipolar electro-coagulation or photodynamic therapy are alternative modalities of treatment.
Fig. 3.38: Barrett's esophagus

Fig. 3.39: Barrett's esophagus with sliding hiatus

hernia
American College of Gastroenterology Surveillance Protocol for Barrett’s Esophagus.

*American Journal of Gastroenterology* 2002; 97: 1888
Chapter 4

MOTILITY DISORDERS OF ESOPHAGUS
Endoscopic features of achalasia cardia include: dilated and tortuous esophagus containing food residue. The LES initially offers resistance to the passage of endoscope but 'gives in' with mild force. The most important aspect of endoscopy, however, is detection of esophageal malignancy consequent upon long standing achalasia. It is also important to exclude 'secondary achalasia' that arises from submucosal infiltration of GE junction by adjacent malignancy. In the latter situation, considerable force is required to negotiate the endoscope across the LES. Once in the stomach, it is mandatory to retroflex and have an optimal view of the GE junction.
Figs 4.3A to D: (A) Barium contrast study showing dilated esophagus and epiphrenic diverticulum (arrow) in a young lady with achalasia cardia. (B) Dilated distal esophagus. (C) Diverticulum (arrow). (D) Non-relaxing LES
Chapter 5

BENIGN GASTRIC ULCER
Based on the location, gastric ulcers are categorized into four types. I: Ulcer located on the lesser curve, II: Associated with duodenal ulcer III: Prepyloric ulcer, IV: Ulcer just below GE junction. Type II and III ulcers are associated with hyperacidity and behave as duodenal ulcer with respect to symptomatology and treatment. Multiple ulcers are seen in association with NSAID use, chronic liver disease, heavy smokers or acute viral infection. Ulcers located high on lesser curvature are likely to be missed during forward passage of the endoscope and best viewed on retroflexion.
Benign Gastric Ulcer

Figs 5.3A to F: (A) Ulcer on the lesser curve (arrow) seen while entering the stomach. (B-D) Close-up view. (E, F) View on retroflexion. The patient presented with hematemesis and melena. Note the ulcer base showing stigmata of recent hemorrhage.
Figs 5.4A to D: Ulcer (arrow) high on lesser curve as seen on retroflexion.

Figs 5.5A to D: Extensive ulceration involving proximal stomach. The patient, a known case of hepatitis, presented with hematemesis.
Benign Gastric Ulcer

Figs. 5.6A and B: Ulcer (arrow) in the body of stomach. Such ulcer is likely to get hidden between the gastric folds. Adequate distention is required for its visualization.

Figs. 5.7A to D: Ulcer in the gastric body at various stages of its appearance during endoscopy. Note the 'flat red spot' suggesting recent episode of bleeding.
Figs 5.8A to F: Endoscopy in an elderly male presenting with pain abdomen and retention vomiting. (A) Retention esophagitis. (B-E) A giant ulcer on the incisura. (F) Another superficial ulcer in the prepyloric region. The pylorus is deformed and narrowed. Note the gastric retention in Figure C.

Figs 5.9A and B: Ulcer on the incisura. Note another small ulcer (arrow) below it.
Fig. 5.10: Ulcer on the incisura having ‘flat red spots’ suggestive of recent hemorrhage

Fig. 5.11: Ulcer on the incisura having ‘flat red spot’

Fig. 5.12: Ulcer (arrow) in the antrum hidden by the blood pool. Note the distorted pylorus (broken arrow)

Fig. 5.13: Ulcer on the incisura covered with acid hematin

Figs 5.14A and B: (A) Giant ulcer on the incisura with adherent clot. (B) Same ulcer one week later. Though partial healing was evident, the patient presented with bleeding recurrence
**Fig. 5.15:** Oozing ulcer in the prepyloric antrum

**Fig. 5.16:** Ulcer in the prepyloric antrum having a ‘visible vessel’

**Fig. 5.17:** Multiple ulcers on the incisura covered with acid hematin

**Fig. 5.18:** Ulcer in the antrum with a clean base

**Figs 5.19A and B:** Giant prepyloric ulcer
Benign Gastric Ulcer

Figs 5.20A and B: Prepyloric ulcers, erosions and pseudodiverticulum

Figs 5.21A and B: Prepyloric ulcer (arrows) hidden in the mucosal folds. Unless the endoscopist is careful, such an ulcer may elude detection

Figs 5.22A and B: Multiple prepyloric ulcers (arrows)
Figs 5.23A to D: (A, B) Ulcers on either side of the pylorus. (C, D) Close-up view of the ulcers.

Figs 5.24A to F: Multiple prepyloric ulcers in a patient with chronic liver disease. (A) Esophageal varices. (B, C) Giant ulcers around pylorus (arrow). (D) Close-up view of the ulcer at 9 o’clock position. (E) Ulcer at 12 o’clock position. (F) Superficial ulcer (arrow) in the duodenal bulb.
Benign Gastric Ulcer

Fig. 5.25: Multiple superficial ulcers in antrum

Fig. 5.26: Ulcer in the prepyloric antrum

Figs 5.27A and B: Prepyloric ulcer

Figs 5.28A to D: (A) Multiple prepyloric ulcers (1, 2, 3). (B-D) Close-up view of the ulcer no.3. Note the adherent clot suggestive of recent bleeding
Figs 5.29A to F: Multiple ulcers (arrows) involving antrum

Figs 5.30A to G: Multiple gastric and duodenal ulcers. (A, B) Ulcers in the antrum. (C) Close-up view of the ulcer at 2 o’clock position. (D) Ulcers in the prepyloric antrum. Note the deformed pylorus and the duodenal ulcer seen through it. (E) The duodenal ulcer appearing black due to presence of acid hematin. (F, G) Close-up view of the duodenal ulcer
**Benign Gastric Ulcer**

Fig. 5.31: Small prepyloric ulcer

Fig. 5.32: Pyloric channel ulcer (arrow)

Figs 5.33A and B: Pyloric channel ulcer with evidence of recent bleed
Figs 5.34A to H: (A) Deformed pylorus (black arrow) and a giant prepyloric ulcer covered with altered blood (white arrow). (B-D): Close-up view of the same ulcer (white arrow) after the blood was cleaned. (E-G) Severe reflux esophagitis in the same patient. Entire esophagus covered with thick exudates. (H) The exudates forming a membrane in the upper esophagus.
**Benign Gastric Ulcer**

**Figs 5.35A to F:** Hour-glass contracture of stomach. (A) Multiple ulcers with cicatization (arrows) causing circumferential narrowing in the gastric body. (B) Ulcers (arrows) are better seen on retroflexion. (C) Antrum, relatively healthy. (D-F) Ulcer healing and scarring (broken arrow) evident after treatment with proton pump inhibitors for eight weeks. Note the mucosal hypertrophy that could have resulted from obstruction as well as hyperacidity. The patient was a chronic smoker.

**Figs 5.36A and B:** Benign gastric outlet obstruction. Scarred and stenotic pylorus consequent upon ulcer healing.
Figs 5.37A and B: Deformed pylorus with prepyloric pseudodiverticulum (arrow). Deformed duodenal bulb can be seen through the pyloric ring.

Figs 5.38A and B: Deformed and narrowed pylorus following ulcer healing.
Chapter 6

CHRONIC DUODENAL ULCER
Fig. 6.1: Duodenitis. Erythematous patches involving duodenal bulb (D1)

Figs 6.2A and B: Duodenitis with superficial ulcer (arrow) in D1

Figs 6.3A and B: Superficial ulcer in the antero-superior wall of D1. The acid-hematin pigmentation suggests recent bleeding
**Chronic Duodenal Ulcer**

**Additional Endoscopic Features of Chronic Duodenal Ulcer**

- Deformity
- Scarring
- Pseudodiverticulum (outpouching of the mucosa)
- Luminal narrowing

Giant duodenal ulcer is defined as an ulcer with a diameter of more than 2 cm.

**Figs 6.4A and B:** Deformed duodenal bulb, multiple superficial ulcers and pseudodiverticuli

**Figs 6.5A and B:** Chronic duodenal ulcer. Ulcer with a clean base present on the anterior wall. Note the deformed bulb and the pseudodiverticuli
Figs 6.6A and B: Deformed duodenal bulb, ulcers (arrows) on the anterior wall and pseudodiverticula

Fig. 6.7: Ulcer (arrows) extending across the pylorus into the posterior wall of duodenum

Fig. 6.8: A healing ulcer on the anterior wall of D1

Figs 6.9A and B: Ulcer on the posterior wall extending from pylorus
**Chronic Duodenal Ulcer**

**Figs 6.10A and B:** A deep ulcer on the anterior wall of D1

**Figs 6.11A and B:** Deformed duodenal bulb and an active ulcer on the anterior wall

**Figs 6.12A and B:** A giant ulcer on the anterior wall of D1
Figs 6.13A and B: "Kissing ulcers" on the superior (white arrow) and inferior wall (black arrow) in D1

Figs 6.14A and B: Giant 'kissing ulcers' in D1. Note the ulcer on the inferior wall (arrow)

Figs 6.15A and B: Multiple ulcers (arrows) in D1
Chronic Duodenal Ulcer

Figs 6.16A and B: Duodenal bulb showing extensive ulceration and Brunner’s gland hyperplasia

Fig. 6.17: Ulcer with surrounding edema in D1

Fig. 6.18: Multiple superficial ulcers in D2

Figs 6.19A and B: Multiple erosions involving D2
Complications of Duodenal Ulcer

- Acute
  - Bleeding
  - Perforation
- Chronic
  - Gastric outlet obstruction
  - Bilio-duodenal fistula/bile duct stricture

Endoscopic Stigmata of Ulcer Bleeding

- High-risk
  - Spurting/pulsatile bleeding
  - Oozing ulcer base
  - Visible vessel
  - Adherent clot
- Low-risk
  - Flat pigmented/red spot
  - Clean ulcer base

Figs 6.20A to C: (A) Spurting bleeding from the ulcer base (arrow) in D1. (B) Bleeding was controlled by injecting adrenaline and saline into the ulcer base. (C) Visible vessel (arrow) in the ulcer base as seen 48 hrs later

Figs 6.21A to D: (A) Actively oozing ulcer in D1. (B) The ulcer base was injected with adrenaline—saline 1: 10,000. (C, D) Ulcer base as seen after endoscopic control of bleeding
Chronic Duodenal Ulcer

Fig. 6.22: "Visible vessel" in the ulcer base

Fig. 6.23: Ulcer and the 'visible vessel' (arrow)

Figs 6.24A and B: 'Visible vessel' in an otherwise clean ulcer base

Figs 6.25A and B: 'Visible vessel' covered with fresh clot
Fig. 6.26: "Visible vessel" accentuated by adherent clot

Fig. 6.27: Ulcer on the anterior wall covered with clot

Figs 6.28A and B: Ulcer on the anterior wall completely covered with a fresh clot

Figs 6.29A and B: (A) A deep ulcer partially covered with clot. (B) Same ulcer eight days later. The clot has been replaced by a 'flat red spot'
Chronic Duodenal Ulcer

Figs 6.30A to D: (A, B) Giant duodenal ulcer with ‘flat red spot’ (arrow) in the center. (C, D) Appearance 24 hours later

Figs 6.31A and B
Suspected perforation is an absolute contraindication for endoscopy. In both the above cases, clinical signs and symptoms were misleading, suggesting acute exacerbation of duodenal ulcer only.
Chronic Duodenal Ulcer

Figs 6.33A to D: Duodenal stenosis: (A) Food residue in antrum and (B) fundus. (C) Deformed duodenal bulb and the pin-hole opening (arrow) seen through pylorus. (D) Duodenal bulb. Note the pseudodiverticulum (broken arrow) and the pin-hole opening (arrow) at its center.

Figs 6.34A to F: (A-C) Deformed duodenum with bilioduodenal fistula (arrow). (D-F) Multiple superficial ulcers (arrow) in the gastric antrum in the same patient. Possibly, the fistula was secondary to duodenal ulcer penetration. Though no active ulcer was noted in D1, the deformed duodenum indicated its earlier existence.
Figs 6.35A to C: (A) Choledochoduodenal fistula. Bile was seen pooling in D1 through a tiny opening at the (arrow) apex. (B) Cannulation and injection of contrast opacified the common bile duct (C) Opacification of proximal biliary tree confirms presence of bilioduodenal fistula. Distal biliary tree did not opacify because of stricture at the fistula site.

Figs 6.36A to F: Choledochoduodenal fistula. (A) Deformed D1. An ulcer (arrow) was seen on the superior wall. (B) Close-up view of the ulcer (arrow) revealed a tiny opening (broken arrow) in its base exuding bile. (C-F) Further inspection of the opening (broken arrow) confirmed it to be bile duct.
Chronic Duodenal Ulcer

Figs 6.37A to L: Giant choledochoduodenal fistula. (A) Deformed D1. Note the pseudodiverticulum (white arrow), an ulcer (broken arrow) and the passage (arrow) to D2. (B) Normal looking D2. (C-E) Pseudodiverticulum (arrow) and closer view of the ulcer (broken arrow). (F) Probing the medial wall of the ulcer led to an oblong opening. (G-I): Close-up view of the opening suggested it to be a part of the bile duct wall having superior and inferior ends (broken white arrows). (J) The bile duct mucosa was quite distinct in its appearance. (K) The superior opening of the bile duct was cannulated and contrast injected. (L) This opacified the proximal biliary tree confirming the presence of a large choledochoduodenal fistula.
Figs 6.38A and B: Deformed D1 with pseudodiverticuli following healed duodenal ulcer

Figs 6.39A and B: Deformed D1 with pseudodiverticuli and superficial ulcers

Figs 6.40A to C: (A) Deformed D1 and pseudodiverticuli seen through pylorus. (B) Closer view of the D1. (C) Close-up view of one of the diverticuli
Chapter 7

GASTROJEJUNOSTOMY
In the past, peptic ulcer surgery accounted for majority of the cases of gastrojejunostomy (GJ). With the sharp decline in the incidence of elective surgery for peptic ulcer, the major indication for GJ, in the present time, is gastric outlet obstruction and partial gastrectomy for various causes. Both early as well late complications of GJ are best evaluated by endoscopy. It is imperative to enter and inspect both afferent as well efferent loop for any pathology.

**Fig. 7.1:** Normal gastrojejunostomy (GJ) stoma. The efferent loop opening is clearly seen (straight arrow).
The afferent loop opening is hidden below the gastric mucosa (curved arrow)

**Fig. 7.2:** Gastrojejunostomy (GJ) stoma

**Fig. 7.3:** GJ stoma along the greater curvature
Figs 7.4A and B: GJ stomal edema. Note the silk suture and the ulceration along the suture line.

Figs 7.5A to D: Stomal edema. (A, B) The GJ stoma is obscured by the two lips of the edematous jejunal mucosa. (C) The afferent loop (arrow head) and the efferent loop (arrow) openings were identified by gentle manipulation. (D) Close-up view of the efferent loop opening.

Endoscopy in the early postoperative period is fraught with the risk of suture line dehiscence. This can be minimized by adhering to the following principles:

- Should be performed by an experienced endoscopist only.
- Use minimal distention and force.
- Proceed only if lumen is visible.
- Avoid entering too much into afferent or efferent loops.
Figs 7.6A and B: Stomal ulcer around a silk suture

Figs 7.7A and B: Stomal ulcer close to efferent loop opening (black arrow). Multiple erosions involving jejunal mucosa (white arrows)

Figs 7.8A and B: Giant stomal ulcer (arrow)
Gastrojejunostomy

Figs 7.9A to E: (A) Small GJ stoma in the body of stomach. (B) A closer look shows presence of an ulcer (arrow). (C, D) Close-up view of the same ulcer on the gastric mucosal aspect of the stoma. (E) The hypertrophic scar on the abdominal wall of the patient. The unusually small scar suggested it to be possibly a case of GJ ‘without’ vagotomy. The patient indeed had both vagi intact for which he underwent laparoscopic truncal vagotomy.
Fig. 7.10: GJ stomal ulcer bleeding. A fresh clot covering the ulcer

Fig. 7.11: Stomal ulcer bleeding. ‘Flat red spot’ on the ulcer surface suggested recent hemorrhage

Figs 7.12A to F: Gastrojejunalcolic fistula. Note the grossly ulcerated stoma and the feculent contents of the efferent loop
Figs 7.13A to D: Gastrojejunocolic fistula. (A-C) Fistulous opening by the side of the GJ stoma containing fecal matter (D) Colonoscopy in the same patient showing two openings in the transverse colon. The lower one was communicating with the stomach. Note staining of the fistulous tract with methylene blue that the patient was made to drink during colonoscopy to confirm the communication.

Figs 7.14A to D: Gastrojejunocolic fistula. (A) Feculent contents in the stomach. Note the efferent loop opening (arrow). (B) A close-up view of the efferent loop opening shows two passages. (C) Entry through one of them (white arrows) led to jejunal lumen. (D) Entry through the other one (black arrow) revealed triangular mucosal folds and fecal matter suggesting colonic lumen, thus confirming presence of gastrojejunocolic fistula.
Endoscopic diagnosis of gastrojejunal fistula is made on the following findings:

- Feculent contents in the gastric lumen
- Grossly ulcerated stoma
- Visualization of colon while manipulating the endoscope through the openings in the stoma.
- Manipulation of the colonoscope in the transverse colon may bring into view jejunal or gastric lumen
- Chromocolonoscopy: With the colonoscope in the transverse colon the patient is asked to drink methylene blue dye. Prompt appearance of the dye in the colon confirms presence of fistula between stomach and colon.

*Tropical Gastroenterology 2001: 22; 221*
Retrograde jejuno gastric intussusception is a relatively uncommon complication of gastrojejunostomy. Efferent loop intussusception is more frequent than afferent loop. The usual presenting features are pain, coffee-ground vomiting and an ill-defined lump in the epigastrium. Diagnosis is confirmed on endoscopy. Early surgery is indicated to prevent strangulation of the jejunal segment.
Figs 7.18A to D: (A, B) Retrograde jejunogastric intussusception. (C) Abdominal ultrasonography showing a circular hyperechoic mass (arrow) inside stomach- the ‘target sign’. (D) Operative photograph showing transmesocolic afferent loop (arrow) and the intussusced efferent loop (broken arrow)

Figs 7.19A and B: Retrograde jejunogastric intussusception. Jejunal loop congested and pregangrenous

Figs 7.20A and B: Retrograde jejunogastric intussusception. Note the gangrenous jejunal loop
Gastrojejunostomy

Figs 7.21A and B: Bile reflux gastritis. Note the sharply demarcated gastric mucosa because of the inflammation

Figs 7.22A to D: (A, B) Inflammatory polyp close to the efferent loop opening in a patient with severe bile reflux gastritis, (C, D) Another polyp close to the afferent loop opening. The patient presented with recurrent anemia
Figs 7.23A and B: Inflammatory polyps at the GJ stomal margin

Figs 7.24A to D: Recurrence of malignancy at the GJ stomal site in a patient who had undergone subtotal gastrectomy for carcinoma two years back
Gastrointestinal stromal tumor (GIST), formerly known as leiomyoma, is the commonest non-epithelial tumor of the GI tract. It arises from the interstitial cells of Cajal, a part of the autonomic nervous system located in the muscular propria. Stomach is the commonest site of its occurrence followed by small bowel, esophagus and colorectum. It commonly presents with bleeding. Mucosal biopsy is usually non-yielding, deeper biopsy may be required for diagnosis. Presence of spindle cell points to the diagnosis of GIST that is further confirmed on positive immunohistology for CD 117 (c-kit). Surgical excision is the preferred treatment. Tumor size < 2 cm and a mitotic index <5/50 HPF suggest low risk tumor.

Figs 8.1A and B: GIST at the lower end of esophagus. This was an incidental finding on endoscopy for dyspepsia

Figs 8.2A to D: ‘Sentinel polyp’. Inflammatory polyp at the GE junction. This is more often an incidental finding on endoscopy
**Benign Tumors**

**Fig. 8.3:** Inflammatory polyp at the GE junction

**Figs 8.4A and B:** Bleeding inflammatory polyp at GE junction

**Figs 8.5A to D:** (A to C) Sliding hiatus hernia with inflammatory mucosal polyp, at various stages of its appearance during endoscopy. (D) The same polyp, prolapsing below GE junction, was seen on retroflexion. Note the central ulceration.
Figs 8.6A and B: GIST in the gastric fundus

Figs 8.7A to D: (A, B) GIST in gastric body. (C) Tumor as seen on gastrotomy. Note the ulcerated center, the site of bleeding in this patient. (D) Bisected tumor after excision
Benign Tumors

Figs 8.8A to D: (A-C) GIST with ulcerated surface in the gastric body. (D) Tumor after excision

Figs 8.9A to D: Multiple adenomatous polyps in: (A) Gastric antrum. (B) Prepyloric region. (C, D) Second part of the duodenum
Fig. 8.10: Multiple inflammatory polyps in fundus

Fig. 8.11: Inflammatory polyps in antrum

Fig. 8.12: Prepyloric submucosal polyp

Fig. 8.13: Prepyloric inflammatory polyp

Figs 8.14A and B: Inflammatory polyp obstructing pylorus
Benign Tumors

Figs 8.15A and B: An inflammatory polyp arising from the pyloric ring. The patient presented with recurrent hematemesis and melena.

Figs 8.16A to F: (A-D) Hamartomatous polyp arising from the pyloric ring. The polyp had multiple fingerlike projections converging on a single stalk. (E) The polyp during surgical excision. (F) The stalk (arrow) after excision. The polyp was an incidental detection in an elderly male who underwent endoscopy for dyspepsia.
Figs 8.17A and B: GIST at the junction of D1 and D2. The patient presented with recurrent melena.

Figs 8.18A and B: (A) GIST in the second part of the duodenum. (B) Pancreateicoduodenectomy specimen showing the ulcerated tumor (broken arrow). Note the position of ampulla of Vater (arrow). The patient, a middle aged male, presented with recurrent episodes of melena.

Fig. 8.19: Submucosal lipoma at the apex of D1

Fig. 8.20: Villous adenoma in D2
Benign Tumors

Figs 8.21A and B: Villous adenoma completely filling D2 lumen. The patient, a middle aged lady presented with anemia and retention vomiting.

Fig. 8.22: Lymphoid hyperplasia in D2

Fig. 8.23: Ulcerated stalk in D2. The patient presented with bleeding ten days after snare polypectomy.
Chapter 9

MALIGNANT TUMORS
Figs 9.1A and B: Ulceroproliferative squamous cell carcinoma in the mid-esophagus

Figs 9.2A and B: Squamous cell carcinoma in the mid-esophagus. Note the impacted food debris (whitish material)

Figs 9.3A and B: Squamous cell, ulcerated tumor
Figs 9.4A to D: Squamous cell carcinoma in mid-esophagus

Figs 9.5A to F: (A-C) Squamous cell carcinoma involving mid-esophagus. (D) Distal tumor free lumen with guide wire in place. (E, F) Self-expandable metal prosthesis across the tumor
Figs 9.6A to C: Self-expandable metal stent across an inoperable tumor in the mid-esophagus.

Figs 9.7A to E: (A, B) Self-expandable metal stent across a malignant tumor. (C-E) The stent coated with food particles, as seen 48 hours later.
Fig. 9.8: Early carcinoma in the distal esophagus

Figs 9.9A and B: Small polyp at the 'Z' line. Clinically the polyp was thought to be inflammatory (Sentinel polyp), biopsy revealed in situ carcinoma
Figs 9.10A to D: (A-C) Adenocarcinoma at the lower end of esophagus. (D) View on retroflexion showing extension of the tumor into stomach.

Figs 9.11A to F: (A-C) Adenocarcinoma at GE junction with accumulated food debris. (D-F) View after removal of the food debris. Increased circumferential involvement distally.
Figs 9.12A to D: (A, B) Adenocarcinoma of GE junction. (C, D) View on retroflexion

Figs 9.13A to C: Ulcerated tumor at GE junction, view on retroflexion

Figs 9.14A to C: Adenocarcinoma GE junction. The tumor extension into stomach as seen on retroflexion
Fig. 9.15: Ulcerated tumor involving gastric fundus

Fig. 9.16: Polypoidal tumor filling the entire fundus

Figs 9.17A and B: Polypoidal adenocarcinoma below GE junction

Figs 9.18A and B: Bleeding polypoidal tumor in fundus
Figs 9.19A and B: (A) Giant ulcer with necrotic base involving lesser curve. Endoscopic biopsy was negative for malignancy; hence, the patient was treated with proton pump inhibitors alone. (B) Partial healing was apparent on repeat endoscopy about six weeks later. But the biopsy from the ulcer margin this time revealed evidence of carcinoma.

Malignant ulcers may also show signs of healing on conservative management. Strict follow-up to demonstrate complete healing and repeated negative biopsies are mandatory to exclude malignancy.

Figs 9.20A and B: Malignant ulcer involving lesser curve and incisura
Figs 9.21A to D: Tumor (arrow) involving distal body and antrum

Figs 9.22A to E: (A-C) Ulceroproliferative growth involving gastric body. (D, E) Circumferential involvement distally
Figs 9.23A to C: Ulcerated tumor starting from incisura to pylorus (arrow)

Figs 9.24A to D: Tumor involving antrum and pylorus
Figs 9.25A to D: Circumferential tumor around pylorus

Figs 9.26A and B: Ulcerated and friable tumor around pylorus
Malignant Tumors

Fig. 9.27: Polypoidal tumor in antrum

Fig. 9.28: Ulcerated tumor around pylorus

Fig. 9.29: Polypoidal tumor around pylorus

Fig. 9.30: Ulcerated tumor with elevated margin around pylorus
Figs 9.31A to D: Linitis plastica: Pangastric involvement

Figs 9.32A and B: Superficial spreading carcinoma involving body and antrum
**Malignant Tumors**

**Figs 9.33A to D:** (A) Tumor in the gastric antrum extending into. (B) Duodenal bulb. (C, D) Synchronous tumor in the second part of duodenum in the same patient.

**Figs 9.34A to D:** Metachronus secondary from a malignant pituitary tumor involving (A, B) Gastric body. (C) Duodenal bulb. (D) Second part of duodenum. The patient, a young male had undergone excision of prolactinoma four years back.
Figs 9.35A to E: Duodenal carcinoma. (A) Normal appearing pyloric mucosa with evidence of submucosal infiltration. (B) Infiltrated pylorus. (C, D) Tumor involving D1. (E) Tumor abruptly stopping at the junction of D1 and D2.

Figs 9.36A and B: Polypoidal tumor (adenocarcinoma) in D1. The patient, a 20 years old male presented with hematemesis.
Fig. 9.37: Ampullary carcinoma

Fig. 9.38: Ampullary bulge produced by a small ampullary tumor

Fig. 9.39: Circumferential involvement of D2 secondary to pancreatic head carcinoma

Fig. 9.40: Duodenal infiltration by renal cell carcinoma
PORTAL HYPERTENSION
**Conn’s Grading**

1. Small varices detectable only on performance of Valsalva maneuver.
2. Small varices (diameter of 1-3 mm) visible without Valsalva maneuver.
3. Varices with diameter of 3-6 mm.
4. Varices > 6 mm in diameter.

*The Journal of Laboratory and Clinical Medicine 1967; 70:442*

**Modified Dagradi Grading**

1. Blue or red varices < 2 mm in diameter.
2. Blue varices 2-3 mm in diameter.
3. Elevated blue veins 3-4 mm in diameter.
4. Tortuous blue varices > 4 mm in diameter, almost meeting in the midline.
5. Grape-like varices occluding the lumen and showing the presence of small cherry-red varices overlying blue-gray varices.

*American Journal of Gastroenterology 1979; 72:395*

**Paquet’s Grading**

1. Small varices without luminal prolapse.
2. Moderate sized varices showing luminal prolapse with minimal obscuring of GE junction.
3. Large varices showing luminal prolapse with substantial obscuring of GE junction.
4. Very large varices completely obscuring the GE junction.

*Endoscopy 1982; 14:4*
Endoscopic recording of esophageal varices
(Japanese Research Society for Portal Hypertension)

1. Fundamental color
   a. White (Cw)
   b. Blue (Cb).

2. Red color signs (RCS):
   (small dilated vessels or microtelangiectasia on varix surface)
   a. Red wale marking (RWM)
   b. Cherry-red spot (CRS)
   c. Hematocystic spot (HCS)
   d. Diffuse redness (DR).

3. Form
   a. Small, straight varices (F1)
   b. Enlarged tortuous varices occupying < 1/3 of lumen (F2)
   c. Large coil-shaped varices occupying >1/3 of lumen (F3).

4. Location (longitudinal extent)
   a. Lower 1/3 (Li)
   b. Mid 1/3 - below tracheal bifurcation (Lm)
   c. Upper 1/3- above tracheal bifurcation (Ls).

5. Adjunctive findings
   a. Erosion (E)

*Gastrointestinal Endoscopy 1981:72; 213*
Fig. 10.8: Three columns of blue varices

Fig. 10.9: ‘Red wale’ markings on varices

Fig. 10.10: ‘Diffuse redness’ on the varices

Fig. 10.11: Secondary varices, i.e. small tortuous collaterals between main variceal columns

Figs 10.12A and B: (A) Fibrin plug (arrow) on a varix just below GE junction indicating the site of rupture. (B) During examination the plug got dislodged and the varix started bleeding actively (arrow)

Fig. 10.14: ‘Pseudo varix’: Focal ectasias in a single column of vein in the esophageal wall

Fig. 10.15: ‘Pseudo varix’: Single column of tortuous vein in mid-esophagus. The GE junction was normal.
Fig. 10.16: Intravariceal sclerotherapy. Sclerosant being injected directly into the varix

Fig. 10.17: Thrombosed varix following sclerotherapy

Fig. 10.18: Thrombosed varix

Fig. 10.19: Thrombosed varix and ulcerated esophageal mucosa

Fig. 10.20: Remnants of thrombosed and obliterated varix

Fig. 10.21: Esophageal ulcer following sclerotherapy
Complications following sclerotherapy:

- **General**
  - Fever
  - Anaphylaxis
  - Septicemia

- **Esophageal**
  - Torn varix
  - Retrosternal pain
  - Dysmotility
  - Dysphagia
  - Ulcers
  - Perforation
  - Stricture
  - Squamous cell carcinoma

- **Pleuropulmonary**
  - Atelectasis
  - Pleural effusion
  - Empyema

- **Distant**
  - Gastric variceal bleeding
  - Bleeding from gastropathy
  - Portal vein periphlebitis
  - Portal vein thrombosis
  - Mesenteric vein thrombosis
Figs 10.27A and B: Endoscopic variceal band ligation (EVL). Ligated varix soon after release of the band

Figs 10.28A and B: Ligated varix

Figs 10.29A and B: Bleeding from ulcerated varix following EVL
Figs 10.30A and B: Thrombosed and ulcerated varix following EVL. (A) The band is still in place. (B) The band has fallen off.

Figs 10.31A to D: Post-EVL ulcers. Punched-out ulcers (arrows) in the esophageal mucosa after sloughing of the thrombosed varices.
### Classification of Gastric Varices

<table>
<thead>
<tr>
<th>Type</th>
<th>Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Varices in the lesser curvature continuous with the esophageal varix.</td>
</tr>
<tr>
<td>2</td>
<td>Fundal varices.</td>
</tr>
<tr>
<td>3</td>
<td>Both lesser curve and fundal varices.</td>
</tr>
</tbody>
</table>

*British Journal of Surgery 1990;77:195*

<table>
<thead>
<tr>
<th>Type</th>
<th>Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Varices appearing as inferior extension of esophageal varices across the squamocolumnar junction.</td>
</tr>
<tr>
<td>2</td>
<td>(Nearly always accompanied by esophageal varices) located in the fundus, which appear to converge towards the cardia.</td>
</tr>
<tr>
<td>3</td>
<td>Varices located in the fundus or body in the absence of esophageal varices and appear unconnected to the cardia.</td>
</tr>
</tbody>
</table>

*British Journal of Surgery 1988;75:195*

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**Fig. 10.32:** Gastroesophageal varix (GOV), Isolated gastric varix (IGV). *Hepatology 1992; 16:1343*

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**Fig. 10.33:** Junctional varix. Varices extending from esophagus across the GE junction

**Fig. 10.34:** Varices just below GE junction
Portal Hypertension

Fig. 10.35: Junctional varix seen on either side of the endoscope

Fig. 10.36: Junctional varix

Fig. 10.37: Varix extending along lesser curve

Fig. 10.38: Varix starting at GE junction and extending along lesser curvature

Fig. 10.39: Diffuse varix below GE junction

Fig. 10.40: Diffuse varix along the lesser curve
Fig. 10.41: Varix below GE junction

Fig. 10.42: Fundal varix

Fig. 10.43: 'White nipple' sign (arrow) on fundal varix suggesting recent bleed

Fig. 10.44: Isolated fundal varix

Large gastric varices appear like 'a bunch of grapes'. Small varices should be differentiated from mucosal folds and prominent submucosal veins. Mucosal folds flatten out on distension. Submucosal veins, unlike varices, do not show dilatation or tortuosity.
Fig. 10.45: Fundal varix

Fig. 10.46: Fundal varix extending from GE junction

Fig. 10.47: Fundal varix appearing like a bunch of grapes

Figs 10.48A and B: (A) Fundal varix being injected with cyanoacrylate glue. (B) The varix appears rounded due to hardening of the glue inside. Some spilled out glue can be seen on the surface of the varix
Figs 10.49A to C: (A) Large fundal varix. (B) Cyanoacrylate glue injection. (C) Solidified varix

Figs 10.50A to C: (A) Ulceration and oozing from the surface of a solidified fundal varix. Cyanoacrylate glue was injected a week earlier. (B) About two months later the ulceration appearing more extensive and the glue extruding out. (C) Continued oozing and further extrusion of the glue 12 days later

Fig. 10.51: Eradicated gastric varix. Scarring and neovascularization in the varix bearing area
Portal hypertensive gastropathy (PHG) is most often an incidental finding in patients of portal hypertension. In <2% patients it may present with significant upper GI bleeding. The endoscopic appearance of the gastric mucosa in PHG is characterized by a beefy red appearance, petechial hemorrhages, red spots, and a mucosal pattern of a white reticular network outlining erythematous central areas (snake skin appearance). Endoscopic biopsies reveal submucosal edema, dilated submucosal veins, mucosal capillaries and venules. Submucosal arterioles have thickened walls with proliferation of endothelial and adventitial elements. The submucosal venules show morphologic features of arterialization. The overall blood flow to the stomach is increased. There are increased submucosal arteriovenous communications resulting in reduction of effective mucosal blood flow.
Figs 10.54A and B: Mucosal appearance in portal hypertensive gastropathy

Figs 10.55A and B: Bleeding portal hypertensive gastropathy
Duodenal varix is found in 0.5-2.5% of patients with portal hypertension. A higher incidence of 43% has been reported in radiological studies (splenoportogram).


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**Figs 10.56A to C:** Duodenal varix. (A) Esophageal varix in a patient presenting with melena. (B, C): Varix with ‘white nipple sign’ was present in D2 signifying the site of bleed

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**Figs 10.57A to C:** Ruptured duodenal varix. Solitary varix in D2 showing ‘white nipple sign’
Chapter 11

CORROSIVE INJURY
In suspected cases of corrosive injury, endoscopy should be performed with utmost care and gentleness following three basic principles—minimal insufflation, avoidance of blind and forceful intubation.

Figs 11.1A to F: Early appearance following acid ingestion. (A) Inflamed pharyngeal mucosa. (B, C) Thin whitish membrane covering esophageal mucosa. (D) Inflamed mucosa beneath the membrane. (E, F) Charred gastric mucosa (contd.)
Figs 11.1G to L: Appearance after four weeks when the patient presented with gastric outlet obstruction. (G) Healed esophageal mucosa. (H) Inflamed gastric mucosa. (I) Prepyloric antrum showing slough and displaced pylorus (arrow). (J) Removal of slough showed inflamed mucosa. (K) Pyloric channel obscured by slough. (L) Barium study showing contracted antropyloric region. The patient was treated with gastrojejunostomy.
Figs 11.2A to E: Acid injury. Early appearance, (A, B) Minimal involvement of esophageal mucosa, (C-E) Necrotic mucosa showing thrombosed vessels in the stomach (contd.)

**Endoscopic Grading of Caustic Injury**

1. Erythema/edema
2a. Friability, hemorrhagic blisters, white exudate, superficial ulcers and erythema
2b. 2a + deep or circumferential ulcers
3a. Small areas of necrosis, brown-black, grayish discoloration, deep ulcers
3b. Extensive necrosis

*Gastrointestinal Endoscopy* 1991; 37:165
Corrosive Injury

(Contd. from previous page)

Figs 11.2F to J: Three weeks after injury. (F) Healed esophageal mucosa. (G) Obliterated fundus of stomach. (H) Inflamed and narrowed body of stomach. (I) Tubular antropyloric region. (J) Barium contrast study of the same patient corroborating the endoscopic findings.
Figs 11.3A to E: Acid injury affecting various parts of the esophagus, appearance in the first 72hrs. (A) Esophagus just distal to cricopharyngeal opening. (B-E) Esophageal mucosa from proximal to distal segment (contd.)
(Contd. from previous page)

**Figs 11.3F to K:** Six weeks after the injury. (F) Tight stricture in the mid-esophagus. (G-J) Stricture opened up by balloon dilatation. (K) Distal end relatively healthy.
**Figs 11.4A to D:** Acid injury. (A) Proximal esophageal mucosa. (B) Distal esophageal mucosa. (C) Thick eschar covering gastric body. (D) Inflamed antral mucosa

**Figs 11.5A to D:** (A, B) Acid injury to esophageal mucosa. (C) Note the hiatus hernia and the relatively healthy gastric mucosa in the hernial sac. (D) Same as seen on retroflexion
Corrosive Injury

Figs 11.6A to F: Chemical burn in a patient who consumed spurious alcohol 24 hrs earlier (A) Superficial burn involving pharynx. (B) Esophagus. (C) Hiatus hernia in the same patient. (D) Retroflex view showing the hiatal sac and the junction of the affected esophageal mucosa and normal gastric mucosa. (E) Close-up view of the same. (F) Multiple superficial ulcers in the gastric body.

Figs 11.7A to E: (A) Corrosive stricture in the mid-esophagus. (B) Guide-wire across the stricture. (C, D) Stricture bearing segment after dilatation. (E) Gastrojejunostomy in the same patient performed earlier for antral stricture.
Figs 11.8A and B: (A) Corrosive stricture involving mid-esophagus. (B) Same after balloon dilatation

Figs 11.9A and B: Corrosive stricture. (A) Before. (B) After dilatation

Fig 11.10: Corrosive stricture

Fig. 11.11: Corrosive stricture after dilatation
Fig. 11.12: Corrosive stricture after dilatation. Note the friable and inflamed mucosa.
Chapter 12

UNCOMMON INFLAMMATORY LESIONS OF UPPER GI TRACT
Esophageal involvement in HIV-infected patients can be:

- Fungal: Candida
- Viral: Cytomegalovirus (CMV), herpes simplex virus (HSV)
- Idiopathic

Figs 12.1A and B: Pharyngeal ulcers in an immunocompromised patient

Figs 12.2A and B: Human immunodeficiency virus (HIV) associated idiopathic ulcer in the mid-esophagus

Figs 12.3A and B: Herpes simplex virus (HSV) ulcers in the mid-esophagus showing minimal involvement
HSV esophagitis commonly presents with acute onset dysphagia, odynophagia and chest pain. On endoscopy, the ulcers appear sharply demarcated having raised margin. Typically known as ‘volcano ulcers’, they may coalesce to cause confluent ulcers.
Figs 12.6A to E: (A to E) Cytomegalovirus (CMV) esophageal ulcers. These are deep ulcers having irregular borders and finger-like projections. (F) The distal esophagus appears normal.
Predisposing Factors for *Candida* Esophagitis

Immunosuppression, diabetes mellitus, corticosteroid therapy, prolonged antibiotics, esophageal obstruction, malignancy.

Gastrointestinal involvement occurs in 3-16% of patients with Behçet's disease. Esophagus and ileocecal region are the two most common sites of involvement.
Figs 12.14A to F: Granulomatous sarcoma of the esophagus. Myeloid cell infiltration of esophageal mucosa in a patient of acute myeloid leukemia. (A) Ulcer in the epiglottis. (B to E) Nodular and ulcerated mucosa with slough involving entire esophagus. (F) No involvement beyond ‘Z’ line

Gastrointestinal Endoscopy 2003; 57:238
Uncommon Inflammatory Lesions of Upper GI Tract

**Figs 12.15A to F:** Drug induced esophagitis. Esophageal ulcers and erosions following doxycycline ingestion the previous day.

**Drugs Commonly Causing Esophageal Injury**

<table>
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<tr>
<th>NSAIDs</th>
<th>Vitamin C</th>
<th>Tetracyclines</th>
<th>Doxycycline</th>
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<td>Tab Potassium chloride</td>
<td>Ferrous Sulfate</td>
<td>Quinine Sulfate</td>
<td>Quinidine</td>
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<td>Zidovudine</td>
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**Figs 12.16A to F:** Acute infectious gastroduodenitis: (A, B) Multiple ulcers in the gastric antrum, (C, D) in D1 and (E, F) in the descending duodenum. The patient presented with acute pain abdomen, vomiting and fever. Mucosal biopsy was nonspecific. Such ulcers were possibly infective in origin. The symptoms resolved on conservative treatment spanning over two weeks.
Figs 12.17A to F: Acute infectious gastrduodenitis. (A) Normal appearing esophagus. (B) Fundus. (C, D) Multiple ulcers in antrum and around pylorus. (E) Ulcers in D1. (F) D2. Patient, a 13 years old boy presented with acute pain abdomen, fever, loose motion and melena. He was empirically treated with oral omeprazole, ciprofloxacin.

Figs 12.17G to L: Endoscopy 12 days later for persistent symptoms showed. (A) Normal esophagus. (H, I) Mucosal edema and ulceration involving fundus and body. Ulcers in (J) Antrum. (K) D1. (L) D2 showing signs of healing.
Endoscopy a week later, for worsening of symptoms and hematemesis, showed diffuse mucosal ulcerations involving (M) fundus, (N, O) body, (P) antrum, (Q) D1 and (R) D2. Mucosal biopsy from the lesions showed non-specific inflammation. Serum gastrin level was normal. The patient was treated with anti- \textit{H. pylori} regimen (Pantoprazole, clarithromycin and metronidazole).

Endoscopy two months later showed slight mucosal irregularity suggestive of healed ulcers in (S) Fundus. (T) Body. (U) Antrum. Complete mucosal healing was observed in (V) D1 and (W) D2.

Clinical Possibilities in the Present Case
- \textit{H. pylori} infection
- Viral infection
- Food allergy
- Crohn’s disease
- HIV infection
Figs 12.18A to D: Extensive ulceration of duodenum. Mucosal biopsy was nonspecific. The patient presented with acute pain abdomen, fever and bloody diarrhea. The symptoms resolved nearly three weeks after conservative management with antibiotics and proton pump inhibitors. The disease possibly represented idiopathic segmental enteritis.

Figs 12.19A and B: Duodenal tuberculosis. Ulcer on the anterior wall at the junction of D1 and D2. The patient presented with low grade fever and weight loss. Biopsy of the ulcer margin showed caseating granuloma.
Figs 12.20A to F: Duodenal tuberculosis. (A, B) Ulcer (arrow) at D1-D2 junction. The patient was started on proton pump inhibitors. (C, D) Repeat endoscopy about 3 wks later showed no signs of healing, instead the ulcer seemed to have worsened. Ulcer biopsy, at this stage, suggested tuberculosis. (E, F): Endoscopy about 1 mo after starting antituberculosis treatment. The ulcer (arrow) showed marked reduction in size. The patient received full course of antituberculosis treatment.
Mallory-Weiss syndrome accounts for 5-10% of all cases of upper gastrointestinal bleeding. The typical presentation is frank hematemesis or blood streaking of vomitus that follows normal bouts of vomiting occurring in the setting of alcoholism, food poisoning or hyperemesis gravidarum. On endoscopy it is characterized by one or more linear mucosal tear involving GE junction. The tear may extend for variable distance onto the GE junction. Bleeding from such lesion/s is usually mild, self limiting and responds to conservative management. Endoscopic intervention, in the form of local adrenaline saline injection, thermal coagulation, hemoclip application or banding may be required in the rare event of continued bleeding.

Fig. 13.1: Linear tear at 10 O’clock position

Fig. 13.2: Linear tears at 2 and 5 O’clock position
Mallory-Weiss Syndrome

Figs 13.3A to F: (A, B) Linear lacerations (arrows) across the GE junction. The patient presented with massive hematemesis following an alcoholic binge. (C, D) Repeat endoscopy 24 hrs later — the altered blood in the lesion has been replaced by whitish slough. Note the sliding hiatus hernia. (E, F) Lacerations extending on to gastric mucosa beyond the hiatal sac as seen on retroflexion.
Figs 13.4A and B: Lacerated esophageal mucosa and submucosal hematoma (arrow) at Z line

Fig. 13.5: A linear tear at the GE junction
Chapter 14

DIEULAFOY'S LESION
Dieulafoy’s lesion (Exulceratio simplex, caliber-persistent artery) is characterized by presence of a thick caliber submucosal arteriole that can cause torrential bleeding. This lesion most commonly occurs in the proximal 6 cm of the stomach. Less commonly it may occur in duodenum and rest of the GI tract. Endoscopic appearance of duodenal Dieulafoy’s lesion ranges from a pin-point dot, clot or tortuous vessel to blood oozing or spurting from normal mucosa. Management includes injection sclerotherapy, monopolar or bipolar heater probe application, laser photocoagulation, band ligation, application of hemoclip or surgical excision.

_Fig. 14.1_ Dieulafoy’s lesion just below GE junction seen on retroflexion

_Figs 14.2A and B_: Actively bleeding Dieulafoy’s lesion in the body of stomach. The bleeding point appeared exaggerated because of the adherent fibrin plug and clot

_Figs 14.3A and B_: Dieulafoy’s lesion in the proximal stomach appearing as tiny protuberance. The patient had massive hematemesis 48 hrs earlier
Dieulafoy's Lesion

Figs 14.4A to D: Dieulafoy's lesion in D1, characterized by punctate oozing (arrow) from an otherwise normal mucosa. Patient, a young lady, presented with recurrent melena. *BMC Gastroenterology* 2003: 3: 2

Figs 14.5A to D: (A) Dieulafoy's lesion, quiescent at the time of examination, is covered with a small clot and blood pool (arrow). Note the surrounding normal mucosa that differentiates it from chronic duodenal ulcer. (B) Repeat examination the following day showed actively bleeding lesion. (C, D) Bleeding was controlled with adrenaline injection.
CHAPTER 15

GASTRIC ANTRAL VASCULAR ECTASIA (GAVE)
Gastric antral vascular ectasia (GAVE) accounts for nearly 4% of variceal upper GI bleeding. The entity commonly occurs in association with chronic liver disease, chronic renal failure, autoimmune connective tissue disorder, bone marrow transplantation, ischemic or valvular heart disease, hypertension, familial Mediterranean anemia and acute myeloid anemia. The pathogenesis of the entity is not clearly understood. The presentation ranges from occult to frank GI bleeding. Two types of lesions have been identified on endoscopy—punctuate or striped. Because of similarity in appearance, the striped variety is also known as 'watermelon' stomach. Though antral region shows predominant involvement, occasionally it may extend to gastric fundus as well. In chronic liver disease, it must be differentiated from portal hypertensive gastropathy as the treatment modalities for both are quite different. Unlike (PHG), reduction in portal pressure has no effect on GAVE. Argonplasma coagulation, laser photocoagulation, heater probe application are the accepted modalities of treatment. Rarely, antrectomy may be required for uncontrolled hemorrhage.

Digestion 2008; 77: 131
Gastric Antral Vascular Ectasia (GAVE)

Figs 15.3A to C: Gastric antral vascular ectasia (Water melon stomach). (A, B) Linear disposition of vascular ectasias, confined to the antrum, resemble the stripes of watermelon. (C) Close-up view of the same.

Figs 15.4A and B: Gastric antral vascular ectasia. (A,B) Linear telangiectasias confined to antrum.
Chapter 16

FOREIGN BODY
Figs 16.1A to E: Coin impacted at the esophageal inlet. (A) Localized to esophageal inlet on chest X-ray. (B to D) Endoscopic appearance and extraction by using 'rat tooth' forceps. (E) Extracted coin.
Foreign Body

Esophageal inlet is the commonest site of foreign body (FB) impaction. Dysphagia, odynophagia, chest pain, excessive salivation are the usual symptoms. Contrary to the common practice, FB extraction should always be performed under general anesthesia. It is our practice to use intravenous propofol anesthesia in adults and intubation anesthesia in pediatric age group. A quiet patient, relaxed cricopharyngeus, secure airway and proper instruments are paramount in successful removal of FB from the upper GI tract.

Management Guidelines

Food bolus in esophagus: Remove urgently if the patient is in distress. Remove electively if the patient is comfortable but do not delay beyond 24 hrs.

FB in esophagus: Endoscopy and removal as early as possible.

Smooth, rounded FB in stomach: Normal passage is expected in 4-6 days but may take up to 4 weeks. Endoscopic removal is recommended for objects more than 2.5 cm in diameter or longer than 6-10 cm or remaining in stomach for more than 4 weeks.

Sharp/pointed objects in stomach: Remove urgently. If it has gone beyond duodenum expectant but watchful management is advocated for any signs of obstruction, perforation or bleeding. Remove if it does not pass out in 72 hrs.

Button battery: Follow if it has gone beyond esophagus. Active removal is indicated for battery more than 2 cm in diameter or if remains for more than 48 hrs in stomach.

Gastrointestinal Endoscopy 2002; 55: 802
Figs 16.5A and B: (A) Denture impacted at cricopharyngeal sphincter held by a snare. (B) After its extraction

Figs 16.6A and B: (A) A fish bone across the mid-esophagus. (B) Mucosal injury at the sites of impaction (arrows) seen after its endoscopic removal

Figs 16.7A and B: (A) A piece of salad (vegetable slice) impacted at esophageal inlet. (B) Dislodged and pushed to distal esophagus by endoscopic manipulation
Figs 16.8A to F: A coin swallowed 2 years back. (A) Localized to upper abdomen (arrow) on X-ray. (B) On endoscopy the coin was found to be in the stomach. Such a rounded object should have, in normal circumstances, passed out in 24-48 hrs period. The persistence of the object in the stomach was due to chronic duodenal ulcer leading to narrowed outlet. (C) Note the deformed duodenal bulb, an active ulcer (arrow) and pseudo-diverticulum (broken arrow). (D, E) The coin was extracted with the help of a Dormia basket. (F) Extracted coin appeared completely blackened due to the prolonged contact with the gastric secretions.
Fig. 16.9: A needle at the junction of body and antrum

Fig. 16.10: A coin in the fundus of stomach

Fig. 16.11: Accidentally swallowed denture in the stomach. Its quadrangular shape and the two spikes (arrows) make its endoscopic extraction extremely difficult and risky. Gastrotomy and removal is our preferred approach for such an object.

Fig. 16.12: A needle in the second part of duodenum. It was held at its proximal end and delivered out along with the endoscope. Holding the needle in such manner is essential to avoid mucosal trauma.
Figs 16.13A to E: (A) Plain X-ray showing multiple needles (arrows) in the GI tract. The patient, a mentally deranged adult, was habitually swallowing sewing needles. (B) Two needles (arrows), the upper one possibly in the descending duodenum. The patient was fortunate to pass out needles without any complications. (C) Needle persisting in the duodenum. (D) Endoscopic view of the same. (E) The needle after its extraction.

Figs 16.14A and B: A bile stained tongue cleaner in descending duodenum. This was accidentally swallowed by the patient about two months back. Endoscopy was done for dyspeptic symptoms.
Chapter 17

TRACHEOESOPHAGEAL FISTULA
Tracheoesophageal fistula (TEF) can be congenital or acquired.

**Etiology of Acquired Fistula**

- **Benign**
  - Pressure necrosis by the cuff of endotracheal/tracheostomy tube
  - Penetrating injury
  - Erosion by impacted foreign body
  - Erosion by caseating mediastinal lymph node
- **Malignant**
  - Locally advanced carcinoma
  - Mediastinal lymphoma after irradiation

Figs 17.1A to E: Benign TEF. Arising from pressure necrosis by the inflated tracheostomy cuff. (A) Bubbling of tracheal secretion through the fistula. (B, C) A rent (arrow) was seen in the cervical esophagus at 12 O’clock position. The transparent tracheostomy cuff is visible through the rent. (D, E) The same rent (arrow) after removal of the tracheostomy tube.
Figs 17.2A and B: Benign TEF consequent upon pressure necrosis by the inflated cuff of tracheostomy tube. The tracheal opening (arrow) was visible upon entering the cervical esophagus. Note the concentric tracheal rings.

Figs 17.3A to F: Malignant TEF. (A, B) Friable tumor involving cervical esophagus. (C, D) Tracheal opening (arrow) was visible while maneuvering the endoscope in the esophagus. (E, F) Closer view revealed the tracheal rings (white arrow).
Figs 17.4A to D: TEF following necrosis of the intervening wall between trachea and esophagus. The patient, a young male had received radiotherapy for mediastinal lymphoma. Note the esophageal lumen (black arrow), the necrotic wall (W) and the trachea (white arrow).
Chapter 18

MISCELLANEOUS
Figs 18.1A and B: (A) Pigmentation involving pharynx, larynx. (B) Esophagus. Incidental finding in an otherwise normal individual.

Figs 18.2A to D: ‘Inlet Patch’. (A) Islands of heterotropic gastric mucosa (arrows) in the proximal esophagus. (B) Patch on the right side. (C, D) Close-up view of the patch on the left side. This is usually an incidental finding and has no clinical significance.
Figs 18.3A and B: 'Inlet patch' (arrow) in the proximal esophagus

Fig. 18.4: Spontaneous esophageal hematoma

Fig. 18.5: Glycogen acanthoma in the esophageal body

Fig. 18.6: Tertiary contraction waves in the esophagus-'feline esophagus'

Fig. 18.7: Tertiary contraction waves and gastric mucosal prolapse at the distal end of esophagus
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Figs 18.9A and B: Esophagogastric anastomosis. Note the residual suture (arrow)

Figs 18.10A to D: Esophagastrectomy. (A) Bile reflux esophagitis. (B-D) Tumor recurrence at esophagastrectomy site. The patient had undergone proximal gastrectomy for a gastroesophageal junction tumor. Note the residual sutures at the anastomotic site
Figs 18.11A and B: Retching gastropathy. (A, B) Congested and ecchymosed gastric mucosa just below the gastroesophageal junction. This has resulted from repeated prolapse through the LES during the act of retching and vomiting.

Figs 18.12A and B: Retching gastropathy. (A, B) Focal ecchymosed gastric mucosa just below gastroesophageal junction.

Fig. 18.13: Retching gastropathy showing signs of healing.

Fig. 18.14: Retching gastropathy.
Fig. 18.15: Angiodysplasia in the proximal gastric mucosa

Fig. 18.16: Ecchymosis produced by nasogastric tube

Figs 18.17A to C: (A, B) Pancreatic pseudocyst producing bulge in the proximal stomach. (C) CT scan image of the same patient showing the thick walled pseudocyst

Figs 18.18A and B: (A) CT scan showing gastric intramural pseudocyst. (B) The edematous antral mucosa. Patient, a known case of recurrent pancreatitis presented with gastric outlet obstruction. Previously, he had undergone percutaneous catheter drainage for retrogastric pseudocyst.

*American Journal of Gastroenterology* 2003; 98: 229
Figs 18.19A to I: (A) Gastric diverticulum in the fundus of stomach (arrow). (B, C) Close-up view of the same. (D) Dilated blood vessels in the wall of the diverticulum. (E) Giant juxtapapillary diverticulum in the same patient. Note the papillary orifice (arrow) in the wall of the diverticulum. (F) Multiple jejunal diverticuli (arrows) were also noted on enteroscopy. Barium contrast study showing the (G) Gastric diverticulum (arrow), (H) Juxtapapillary diverticulum (arrow), (I) Jejunal diverticuli. The patient presented with massive lower GI bleeding.
Fig. 18.20: Percutaneous endoscopic gastrostomy (PEG) tube in the stomach

Fig. 18.21: PEG tube, indigenously prepared from Foley’s catheter

Figs 18.22A to E: (A to C) True diverticulum (arrows) in the superior wall of D1. (D, E) Close-up view of the same. Note the healthy and normal appearing mucosa inside the diverticulum

Fig. 18.23: Minor papilla in D1. The umbilicated appearance is quite typical
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Fig. 18.26: Multiple hookworms in D1

Fig. 18.27: Hookworm in D1
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Figs 18.29A and B: (A) Endoscopic retrograde cholangiogram showing a roundworm in the proximal biliary tree. (B) The worm was extracted after grasping it with an endoscopic ‘rat tooth’ forceps.

Fig. 18.30: Roundworm across a choledochoduodenostomy stoma. Note the bile duct lumen (B) and the duodenum (D).

Fig. 18.31: Roundworm in D3.
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Figs 18.33A to C: (A) Cholecystoduodenal fistula. Active pus discharge was noted in D1. (B) CT scan showing air inside distended gallbladder and (C) in the intrahepatic biliary radicles. Patient, a known case of gallstone disease was admitted with acute cholecystitis. Resolution of his symptom coincided with the formation of such fistula, a fact not so well described in the literature.
Figs 18.34A to F: Bouveret’s syndrome: (A, B) An external bulge was noted in the antropyloric region. (C) A large size gallstone was found impacted in the D1. (D) Endoscopy performed 48 hrs later showed pus discharge from the bulge. (E) The gallstone had passed down, the site of impaction showing mucosal irregularity. (F) Postbulbar duodenum. The patient presented with gastric outlet obstruction that resolved spontaneously with the passage of the offending stone.

Indian Journal of Gastroenterology 2004; 23: 109

Figs 18.35A and B: A pigment stone impacted at the ampulla of Vater
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