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British Society of Gastroenterology and Association of Upper Gastrointestinal Surgeons of Great Britain and Ireland guidance on best practice for upper gastrointestinal endoscopy

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ABSTRACT

Background National root cause analysis of post-endoscopy upper gastrointestinal (UGI) cancer in England has found wide variations in UGI endoscopy quality. This guidance aims to provide a practical UGI endoscopy guide to improve procedural quality, optimise early malignant and pre-malignant lesion detection and common pathology managment.

Methods An initial consensus document was drafted in 2023 by the Scottish National Endoscopy Training Programme. A Guidance Development Group including endoscopy academy and regional endoscopy leads, devolved nation, JAG, AUGIS and BSG representatives was subsequently convened to adapt the document for UK-wide use. Targeted literature reviews were undertaken to provide evidence where available and recommendations were refined through expert consensus.

Results High quality UGI examination is facilitated by closed mouth local anaesthetic spray application and combined sedation with opioids and benzodiazepines when sedation is needed. Mucosal cleansing with Simethicone and N-Acetyl Cysteine is recommended. Systematic inspection is recommended during diagnostic UGI endoscopy: first full oesophageal assessment using both white light and digital chromoendoscopy (facilitates squamous neoplasia detection); then complete gastric examination with white light in retroflexion and antegrade, with virtual chromendoscopy for any focal abnormality; and finally duodenal examination. In Barrett's oesophagus, following mucosal cleansing and white light examination, virtual chromoendoscopy and acetic acid enhance dysplasia detection. When gastric atrophy or intestinal metaplasia are suspected, virtual chromoendoscopy with targeted biopsies, if a focal lesion is present, and Sydney protocol biopsies to establish the extent of atrophy/metaplasia are recommended.

Conclusions Optimising mucosal visualisation through sedation when appropriate, mucosal cleansing and chromoendoscopy enhances recognition of pre-malignant and early maligant lesions in the oesophagus and stomach and is recommended.

The initial version of the following consensus document was written by members of the academy of the Scottish National Endoscopy Training Programme in 2023. The document was then shared at a meeting of representatives of English endoscopy academies, National Health Service England regional leads and their equivalents in the devolved nations and

representatives of Joint Advisory Group for Gastrointestinal Endoscopy (JAG), Association of Upper Gastrointestinal Surgeons of Great Britain and Ireland (AUGIS) and British Society of Gastroenterology (BSG) in December 2023 at a BSG Upper GI endoscopy quality improvement meeting arranged to improve standards of upper gastrointestinal (UGI) endoscopy in the UK. Given the practical benefit of a UK consensus document on best practice for UGI endoscopy and with agreement from the Clinical Services and Standards Committee of the BSG, a subgroup from the meeting was asked to form a Guidance Development Group (GDG), adapt the document for use throughout the UK and provide expert consensus on the content.

Focused literature searches were undertaken for each area covered by the guidance to provide evidence when available for the best practice described and the text refined by consensus among the GDG members. It is intended as a practical guide to performing upper GI endoscopy and the management of common or important pathologies seen during upper GI endoscopy.

The final version of the guidance was submitted to the Clinical Services and Standards Committee of the BSG for independent peer review. Following responses to peer review to the satisfaction of the BSG Guidelines Lead, the guidance was accepted by the BSG and subsequently by AUGIS, following internal review within AUGIS.

BSG guidance represents a consensus of best practice based on the available evidence at the time of preparation. They may not apply in all situations and should be interpreted in the light of specific clinical situations and resource availability. Further controlled clinical studies may be needed to clarify aspects of these statements, and revision may be necessary as new data appear. Clinical considerations may justify a course of action at variance to these recommendations, but we suggest that the reasons for this are documented in the medical record. BSG guidelines and guidance are intended to be an educational device to provide information that may assist in providing care to patients. They are not rules and should not be construed as establishing a legal standard of care or as encouraging, advocating, requiring or discouraging any particular treatment.

OPTIMAL UPPER GI ENDOSCOPY

High quality UGI endoscopy requires optimal practice at each stage from pre-admission, the procedure itself to report writing. The procedure is less technically challenging than, for example, colonoscopy, but the range and subtlety of pathology encountered is much greater. High-quality UGI endoscopy, as described below, maximises the opportunity to detect important lesions, including premalignant and early malignant lesions, and minimise post endoscopy UGI cancer.

Key points

- Consent to UGI endoscopy should be obtained according to the process outlined in the BSG consent guidance.[1]

Preadmission

- Written and online resources that inform patients about the process and preparation should be available.[2]
- Appropriate fasting—for food, including milk, for 6 hours prior to the procedure. Water can be taken up to 2 hours before the procedure.[3 4]
- Continue proton pump inhibitors (PPIs).[3] A small group of patients may require proton pump inhibitior withdrawal, for example, possible eosinophilic oesophagitis[5] or for *Helicobacter pylori* testing.[6] If *H. pylori* status is relevant to a patient's endoscopy findings or previous history and rapid urease testing is negative in a patient already on a PPI or who has had antibiotics in the past month, we suggest performing stool antigen testing post-procedure after a 2-week PPI wash-out period.
- For diagnostic upper endoscopy, continue antiplatelet therapy. Non-vitamin K oral antocoagulants (NOAC)/direct oral anticoagulants (DOAC) should be omitted on the morning of the procedure to allow biopsies to be taken if necessary. Warfarin should be continued, but the international normalised ratio (INR) should be checked in the week before the endoscopy, to ensure it is within therapeutic range to allow for biopsies to be taken if needed.[7]

Preprocedure

- Patients need reassurance as many are anxious about the prospect of endoscopy: individual and team endoscopy non-technical skills (ENTS) are important in this.
- *≥15 min prior to the procedure*[8] the following is recommended provided that there is no swallowing impairment:
  - A mixture of mucolytic and defoaming agent should be used to improve mucosal visualisation[9]: 100 mL of water with 2 mL of N-acetylcysteine (200 mg/mL; Parvolex, Celltech, UK or one 600 mg dissolvable tablet of NACSYS, Alturix, UK) and 0.5 mL of simethicone (40 mg/mL; Infacol, Forest Laboratories, UK).[10] If this is not available, the following is recommended: 100 mL of water containing 3 mL of simethicone (Infacol).[8] This is not as effective in removing mucus from the oesophagus or stomach but is better than no preprocedure preparation.
- Intravenous access (cannula) is recommended before entering the endoscopy room for all patients who may need sedation or Buscopan.
- The history, medication, previous procedure(s) and pathology information, when available, should be reviewed by the endoscopist.
- Consider asking other risk stratification questions[10]: family history of gastro-oesophageal cancer, smoking, alcohol, previous head and neck or lung cancer (if not already covered in preprocedure paperwork).

Procedure equipment

- High-definition endoscopy equipment with virtual chromoendoscopy.[11]
- A foot pump water irrigation system should be available for mucosal cleansing.
- N-acetyl cysteine (Parvolex) and simethicone (Infacol) should be available for mucosal cleansing during the procedure.[10]
- Buscopan (10–20 mg intravenously) should be available and considered if adequate views are not possible due to gut motility.[9 10]
- Acetic acid (2.5 or 3%) should be available for Barrett's oesophagus chromoendoscopy via a spray catheter.[12]
- A distal attachment cap should be available in the department if needed for assessment of lesions,[13] for example, the gastro-oesophageal junction (GOJ) where the view can be difficult, or for endotherapy.

Sedation

- Non-pharmacological interventions, such as auditory or visual distraction, to reduce patient anxiety are recommended.[14]
- There should be a process of shared decision making when choosing local anaesthetic throat spray and sedation options.[14]
- For a high-quality procedure, the majority of patients will require sedation using a combination of opioid and benzodiazepine.[15] This is particularly important for therapeutic and high-risk surveillance/assessment (eg, Barrett's oesophagus). The drugs act synergistically and doses should be adjusted accordingly.
- Local anaesthetic throat spray with sedation is recommended where there is a low risk of aspiration.[14] We recommend avoiding throat spray in those at increased risk of aspiration,[14] for example, patients with possible upper GI tract obstruction or patients with acute stroke.
- Patients undergoing endoscopy regardless of sedation should have monitoring of pulse, oxygen saturations, blood pressure (BP) and respiratory rate before and after the procedure as a minimum.[14] For those sedated, intraprocedural BP and oxygen monitoring is required.[16] BP monitoring is recommended to be carried out every 5 min.[17]
- Patients with renal or liver disease or American Society of Anesthesiologists (ASA) ≥3 are recommended to usually receive ≤50% of the sedation doses used in healthy patients.[14]
- Fentanyl is the preferred opioid for sedation. It has a 1–2 min onset to maximum effect[17] and also suppresses the gag reflex.
- To minimise sedation-related complications, endoscopists should consider patients' comorbidities, ASA score and if over the age of 65, their clinical frailty scale.[18]

Topical local anaesthetic technique

Xylocaine (lidocaine hydrochloride) 10%, can be used up to 20 sprays.[19] Application using a closed mouth method will cover the base of the tongue, uvula, palate, posterior pharyngeal wall, palatopharyngeal and palatoglossal folds. This will improve patient tolerance, reduce anxiety and cough.[20] Be aware that a side effect of this is increased salivation, so suction should be available. The patient should have an absorbable pad available to catch saliva during the procedure.

Technique:

- Warn the patient about the sensation of swelling or choking in the throat.
- Angle the spray nozzle to one side of the oral cavity at the level of the back of the tongue with the mouth open.
- Close the lips.
- Apply 10 pumps, remove the nozzle from the mouth, gargle and swallow.
- Angle the nozzle to the other side. Apply another 10 pumps, gargle and swallow.
- Wait 2–3 min.

Test the endoscope

If fluid leaks from the tip of the endoscope or there is a strobe effect on the screen, the air-water valve is probably leaking and should be replaced. A leaking valve will cause airway irritation and coughing. Ensure adequate air insufflation, suction, lens washing and water jet function, together with a clear colour endoscope view on the screen, before intubation.

Before introducing the endoscope

Empty the suction channel of any residual fluid by applying the aspiration button for 2–3 s. This minimises fluid entering the pharynx when air insufflation is applied.

Lubricate the endoscope by applying a thin smear of lubricating jelly avoiding the camera lens at the tip of the scope.

The patient's head should be slightly elevated and suction available to minimise aspiration risk. The patient's head in the neutral position allows scope alignment with the oesophagus, that is, do not put chin to chest. Tip the face slightly into the pillow, which allows any secretions to come to the front of the mouth, away from the airway.

Intubation

With the endoscope aligned with the patient's oesophagus, hold the shaft 25 cm from the tip and place the tip on the mouth guard. This should allow the endoscope to cross the cricopharyngeus into the oesophagus before the endoscopist needs to adjust their hand position on the endoscope shaft. Avoid shining the light of the endoscope into the patient's eyes. The tongue will be at 12 o'clock and the endoscope should be in the midline. Slowly advance with a tip-up movement passing to the pyriform fossa. The vocal cords should be visualised and photographed if possible.

Advance the endoscope on either the right or left side of the pyiform fossa, with upward deflection,

passing into the upper oesophagus under direct vision. Trying to swallow may cause constriction of the cricopharyngeus and may make intubation more difficult. Instead, ask the patient to take deep breaths in and out to relax the cricopharyngeus. If resistance to advancing the endoscope or a 'red out', pull back and reorient the endoscope.

Oesophagus

Once in the oesophagus, pause to let the patient accommodate to the presence of the endoscope and insert a small amount of air to obtain a luminal view.

The frequency of significant (neoplastic) pathology is highest in the oesophagus, then the stomach and finally the duodenum. More time is required to inspect the areas of greatest risk. The patient's symptoms are also relevant, for example, dysphagia should lead to a very careful inspection of the oesophagus, GOJ and fundus.

Clean the oesophageal mucosa if there are any bubbles or mucus,[11] being careful to avoid aspiration. Inspect the whole oesophageal mucosa in white light and a second time with digital chromoendoscopy (narrow band imaging/blue light imaging/iSCAN), including any lesions detected. This improves the detection of squamous dysplasia and early neoplasia (the most commonly missed upper GI cancer during endoscopy) and inlet patches.[21] Each appears darker than the background mucosa. The vascular and pit pattern and mucosal colour changes on digital chromoendoscopy allow an increased detection of dysplasia.[22 23] Near focus or magnification can assist further. A distal cap attachment may also help, particularly at the GOJ. Take images of the upper and lower oesophagus and of any pathology noted.

Gastro-oesophageal junction

Identify the GOJ before passing through. This approach allows for a thorough assessment before possible endoscope trauma at the GOJ, which can make it harder to see subtle changes. The GOJ is currently defined as the top of the gastric folds with only a moderately distended oesophagus (figure 1). Overdistending the oesophagus will obscure the top of the gastric folds and may create the impression of Barrett's oesophagus. This will also be the squamocolumnar junction in a normal oesophagus (Z line). Note the level of diaphragmatic pinch and the presence of a hiatus hernia (best assessed in retroflexion in the stomach). Measurements of the distance of the GOJ from the incisors, the level of the diaphragm and length of a hiatus hernia should be taken on withdrawal.

The Kyoto international consensus aims to change the definition of the GOJ to the bottom of the palisade vessels, instead of the top of the gastric folds.[24] These are the vessels that run vertically in the distal end of the oesophagus. However, these vessels are usually not visible in the presence of inflammation at the GOJ (eg, reflux oesophagitis)—therefore the proximal limit of the gastric folds is often a better marker.

Take a photograph of the GOJ from above. Complete oesophageal inspection with white light and digital chromoendoscopy and take photographs before traversing into the stomach. The highest-risk area for dysplasia/cancer at the GOJ is from 12 to 3 on the clock face.[13]

Stomach

Pass through the GOJ into the stomach; the initial movement is usually a small amount of anticlockwise torque with tip-up. Then insert a small amount of air and aspirate any fluid. Advance using clockwise torque to the antrum, taking care to see the lesser curve in anteversion. The stomach examination should normally be completed before passing through the pylorus. Neoplastic disease is more likely in the stomach than in the duodenum,[25 26] and once the pylorus is traversed, air enters the small intestine, increasing patient discomfort and reducing tolerance. Retroflex the endoscope by advancing towards the pylorus and bringing the tip up until the incisura comes into view. Inspect and photograph the incisura.

Then insert the instrument until the tip is beyond the incisura and rotate the instrument to achieve retrograde views of the stomach. Withdraw the endoscope and rotate to achieve a 360-degree view of the upper stomach, inspecting the lesser curve in particular as withdrawing. The lesser curve is

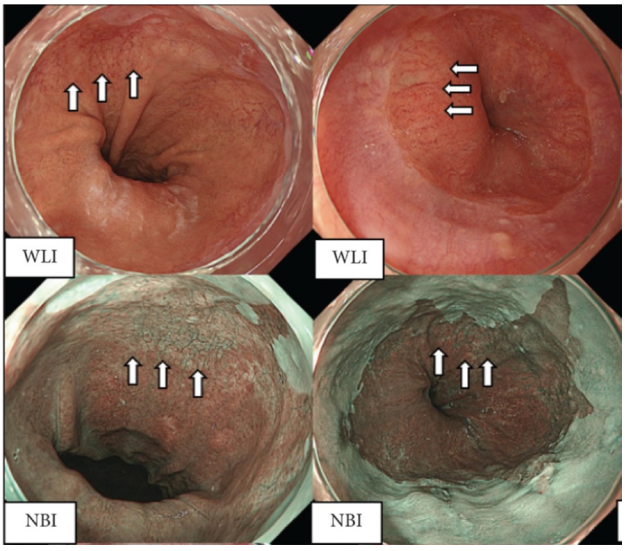

Figure 1 Palisade blood vessels at GOJ. Arrows indicate palisade vessels within Barrett's oesophagus. Reproduced from Ueda *et al*, 'Improved visibility of palisade vessels within Barrett's esophagus using red dichromatic imaging: a retrospective cross-sectional study in Japan', published in *Clinical Endoscopy*, under a Creative Commons Attribution Non-Commercial Licence 4.0 [25,26]. Reprinted with permission from the Korean Society of Gastrointestinal Endoscopy (Clin Endosc. 2025;58(2):269-277). GOJ, gastro-oesophageal junction; NBI, narrow-band imaging; WLI, white-light endoscopy.

the area with the highest risk of gastric atrophy, gastric cancer and benign gastric ulcers. Withdraw the endoscope further and rotate to achieve a 360-degree view of the GOJ in retroflexion. This careful assessment is required to avoid missing pathology behind the shaft of endoscope at the GOJ. Take a photograph. Do not withdraw the retroflexed endoscope into the tubular oesophagus.

Gently release the retroflexion, inspect the stomach in anteversion, white light and virtual chromoendoscopy. Photograph the body and antrum.

Duodenum

Traverse the pylorus taking care not to impact the tip on the duodenal mucosa. Insufflate and pause to fully visualise the duodenal bulb before passing to D2, noting the appearances before any potential endoscope trauma. Photograph D1. Apply more clockwise torque with tip up to enter D2. Photograph D2. Slowly withdraw with an anticlockwise torque when passing from D2 to D1 while also bringing the tip down. On re-entering the stomach aspirate to deflate the stomach prior to entering the oesophagus. Slowly withdraw, completing inspection. NB: If planning to reintroduce the endoscope (eg, for banding), empty the stomach and oesophagus of fluid as withdrawing.

A minimum of 7 minutes total procedure time is recommended.[4 27–29] 10 anatomical landmark photographs are recommended as a minimum: proximal oesophagus (figure 2 Image 1), distal oesophagus (Image 2), Z-line and diaphragm indentation (Image 3), cardia and fundus in retroflexion (Image 4), corpus in forward view including lesser curvature (Image 5), corpus in retroflex view including greater curvature (Image 6), angulus in partial inversion (Image 7), antrum (Image 8), duodenal bulb (Image 9) and second part of duodenum (Image 10). The aim of photo documentation is to: show crucial landmarks, document the extent of examination, and indicate the quality of mucosal views and cleansing.[30]

A comprehensive instructional article that details each stage of UGI endoscopy, including annotated photographs, demonstrating endoscope insertion and navigation through anatomical landmarks from the oral cavity to the duodenum can be accessed via Doi: 10.3748/wjg.v21.i3.759

Report writing

Electronic records systems provide standardised, systematic, universally descriptive language in endoscopy that is essential for effective communication between endoscopists and clinicians. Endoscopy reporting systems should

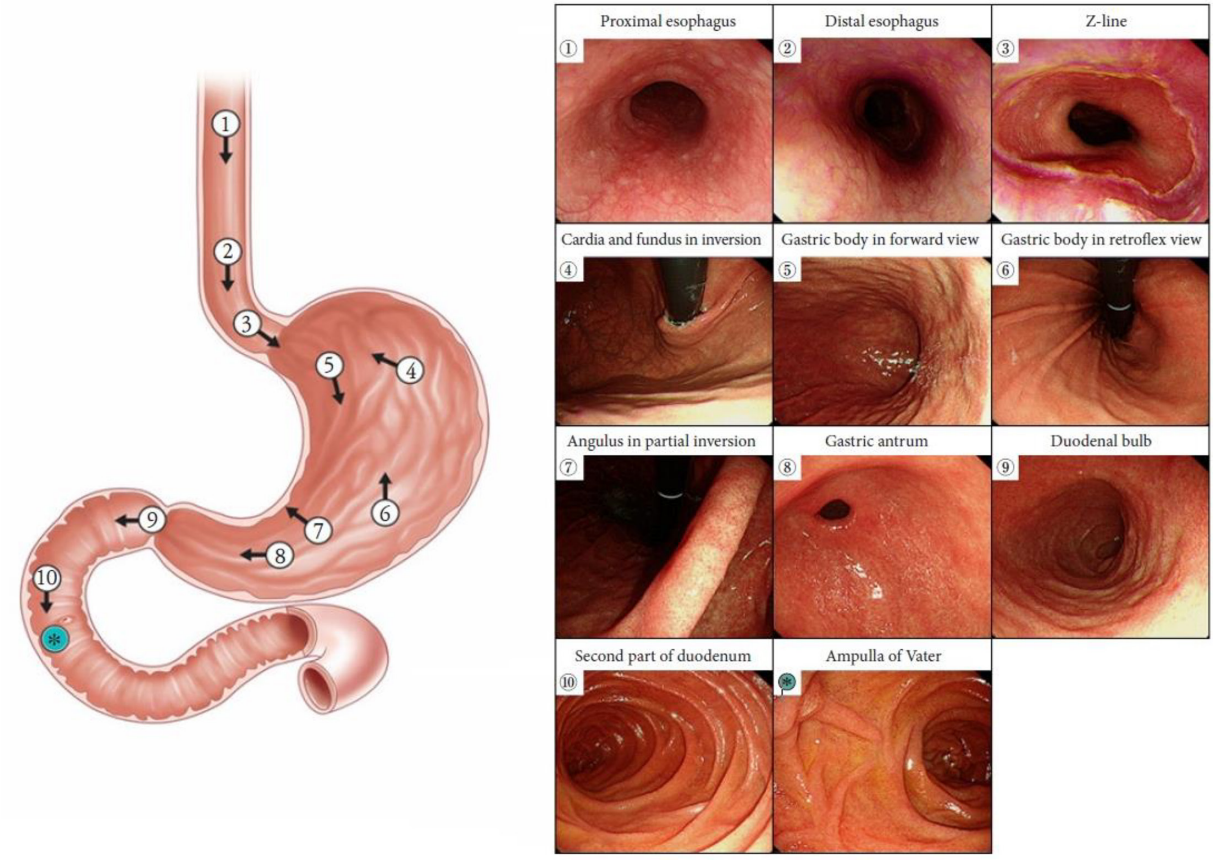

Figure 2 Photo-documentation in normal endoscopic examination. Reproduced from Kim SY *et al*, 'Quality indicators in esophagogastroduodenoscopy', published in *Clinical Endoscopy*, under a Creative Commons Attribution Non-Commercial Licence 4.0.[109 110] Reprinted with permission from the Korean Society of Gastrointestinal Endoscopy (Clin Endosc. 2022;55(3):319-331).

restrict the use of free-text entry to a minimum and be based mainly on structured data entry.[31]

The following information should be included for UGI endoscopy:

- Patient tolerance of the endoscopy and whether this impacted on the quality of mucosal views.
- Extent of examination and reason for incomplete examination, if appropriate.
- Describe the quality of cleansing/visualisation including measures taken and their results (flushing modality—syringe vs foot pump), position changes and mechanical removal).[30]
- Technical/procedural aspects should be documented (eg, Barrett's inspection time, total examination and withdrawal times, any specific technical problems encountered during the procedure—and their resolution, to facilitate future repeat examinations).[30]
- Describe specific negative findings that may be of relevance in the context of the indication for endoscopy, for example, 'no endoscopic oesophagitis', 'no hiatal hernia' and 'no signs of recent bleeding'.
- Indication for any biopsies.
- Recommendation in terms of surveillance, follow-up and restarting anticoagulation if appropriate.

ENDOSCOPIC NON-TECHNICAL SKILLS

The 2004 Confidential Enquiry into Patient Outcomes and Death report (Scoping our practice) investigated deaths of 1818 patients within 30-days of therapeutic endoscopy in the UK.[32] Many recommendations highlighted failings in team-work and non-technical skills, which affected procedural planning, monitoring of patient during the procedure and the administering of sedation safely.[33] Non-technical skills are cognitive and social skills that influence quality and safety outcomes.[34]

JAG recognises the importance of ENTS to safety in endoscopy and has adopted the ENTS framework into Direct Observation of Procedural Skills and developed ENTS simulation courses. The ENTS framework adopted by JAG should be followed during endoscopy.[35]

Category	Element
Communication and teamwork	Maintains clear communication
	Gives and receives knowledge and information in a clear and timely fashion
	Ensures team and endoscopist working together
	Ensures patient is centre of procedure, ensures safety and comfort
	Clear communication of results and management plan with patient/carers
Situation awareness	Procedure carried out with respect and dignity
	Continuous evaluation of patient condition
	Ensures lack of distractions and maintains concentration, particularly during difficult situations
	Intraprocedural changes to scope setup monitored and rechecked
Leadership	Provides emotional and cognitive support to team members by tailoring leadership and teaching style appropriately
	Supports safety and quality by adhering to current protocols and codes of clinical practice
	Adopts a calm and controlled demeanour when under pressure, using all resources to maintain control of situation and taking responsibility for patient outcome
Judgement and decision making	Considers options and possible courses of action to solve an issue or problem, including assessment of risk or benefit
	Communicates decisions and actions to team members prior to implementation
	Reviews outcomes of procedure or options for dealing with problems
	Reflects on issues and institutes changes to improve practice

EARLY UPPER GI MALIGNANT LESION RECOGNITION

Adequate mucosal visualisation is key to early malignant lesion recognition and should be achieved by a combination of adequate air insufflation, aspiration and the use of mucosal cleansing techniques.[2]

Key points

- High-definition endoscopes with magnification and image enhancement should be used.[11 36]
- White light for lesion recognition and morphology (Paris classification).[10]
- Image enhancement for:
  - Lesion demarcation line.
  - Dysplasia recognition—seen as irregularity to loss of the normal vascularity (eg, intrapapillary capillary loops (IPCLs) in the oesophageal squamous mucosa) and the normal gland structures. Areas of dysplasia will appear darker on virtual chromoendoscopy due to increased vascularity.[37]
- Enhanced imaging requires the mucosa to be clean and bubble free and no blood to be present (appears dark on enhanced imaging).
- Lesions should be imaged prior to biopsy or other intervention.[2 38]
- Strictures should be biopsied prior to intervention.[2]

Further guidance on biopsy sampling during UGI endoscopy can be accessed via doi:10.1136/flgastro-2025-103316.

OESOPHAGEAL CANCER

A recent English study of 106 557 patients found that 8.5% of them had undergone endoscopy in the preceding

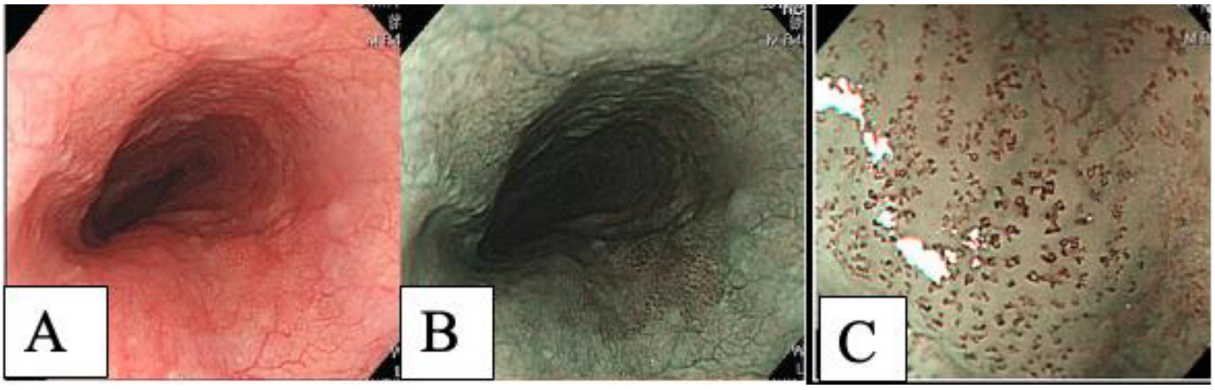

Figure 3 Oesophageal squamous cell carcinoma. (A) An uneven hyperaemic mucosa under white-light endoscopy. (B,C) Brownish discolouration of mucosa with abnormal microvascular pattern under narrow band imaging. Adapted from Chung CS *et al*, 'Long term outcome of routine image-enhanced endoscopy in newly diagnosed head and neck cancer: a prospective study of 145 patients', published in *Scientific Reports* 6, Article number: 29573 (2016) under Creative Commons Attribution 4. [50,130].

3 years that did not diagnose cancer. 64% had potentially missed oesophageal cancer.[39]

Key points

- Oesophageal adenocarcinoma (OAC) accounts for two-thirds of oesophageal cancers. Risk factors include obesity, gastro-oesophageal reflux and Barrett's,[40] and it originates usually in the lower third of the oesophagus.[41]
- Oesophageal squamous cell carcinoma (OSCC) risk factors include alcohol, smoking and head and neck cancer and it originates usually in the upper two-thirds of the oesophagus.[41]
- Pharynx-localised dysphagia is more likely to be a referred symptom of structural oesophageal disease, including cancer, than a primary symptom of structural pharyngeal disease.[42]
- Inspect the oesophagus initially under white light and look for areas of sharp delineation or inhomogeneity of colour distribution.[43] Superficial OSCC appears as an uneven and reddish lesion possibly with an unclear boundary and sometimes a white coating on the mucosal surface with a less visible vascular network.[44] Then inspect using image enhancement. Normal oesophageal mucosa will appear green in colour, while in the presence of squamous neoplastic lesions there will be brownish discolouration (figure 3) and abnormal IPCLs (figure 4).[45]
- Oesophageal cancers should be described in terms of size, site as distance from the incisors for the upper and lower border and Paris classification: flat (IIb), protruding (Is) or elevated (IIa), ulcerative (IIc) or diffusely infiltrative[46] (figure 5) clockface position should be used for non-circumferential lesions.
- Advanced oesophageal cancers should have at least eight biopsies taken but only 1–2 targeted biopsies from potential early cancers to avoid compromising potential later endoscopic resection.[38]
- If an oesophageal ulcer appears malignant, then at least eight biopsies are required.[38]
- On-line learning to help identify early Barrett's neoplasia: BORN project https://iwgco.net/born-program/born-presentation/.

BARRETT'S OESOPHAGUS

Barrett's oesophagus is columnar lined epithelium extending above the GOJ for ≥1 cm confirmed histologically by the presence of intestinal metaplasia.[47] It is associated with an increased risk of OAC.

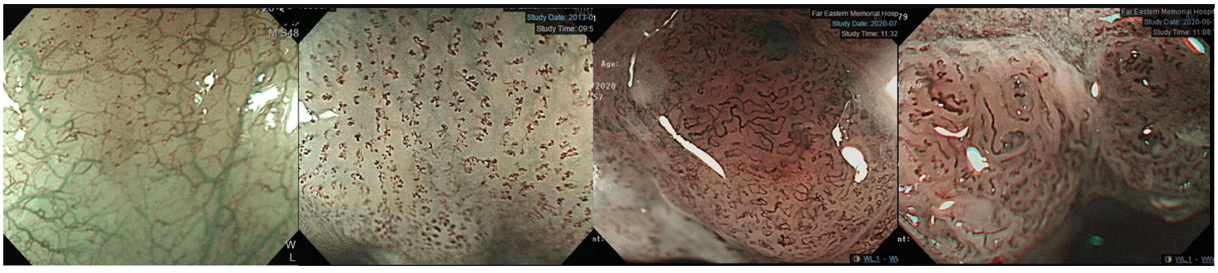

Figure 4 JES classification of microvessel morphology of IPCL. From left to right: JES type A, normal IPCL without irregularity. JES type B1, abnormal microvessels with severe irregularity, meandering calibre or highly dilated proliferative abnormal vessels with a loop-like formation. JES type B2, abnormal microvessels with severe irregularity, meandering calibres or highly dilated proliferative abnormal vessels without a loop-like formation. JES type B3, highly dilated microvessels with three times as many calibres as usual type B2 vessels. Reproduced from Chung CS *et al* 'Endoscopic Screening for Second Primary Tumors of the Esophagus Among Head and Neck Cancer Patients', published in *Frontline Oncology* 2022 Jun 7:12:906125, under creative commons licence 4.0 [50,131]. IPCL, intraepithelial papillary capillary loop; JES, Japanese Esophageal Society.

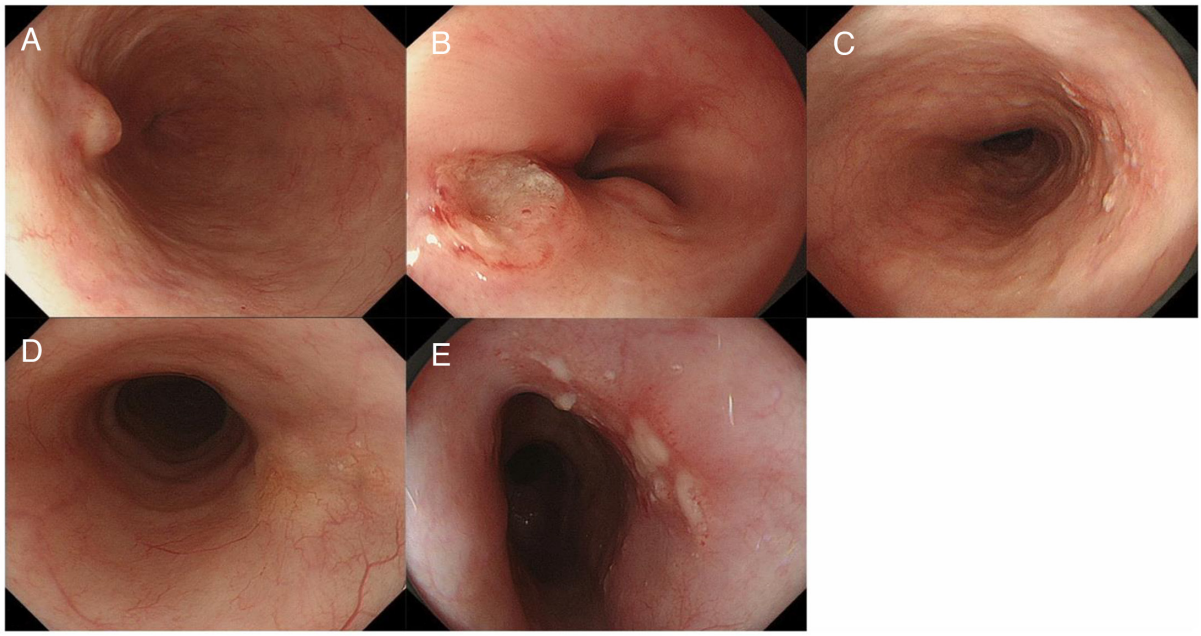

Figure 5 Representative images of endoscopic findings. (A) Protrusion. (B) Excavation. (C) Unevenness. (D) Submucosal tumor-like appearance. (E) Erosion. Various appearances of oesophageal squamous cell carcinoma. Reproduced from Tani Y *et al*, 'Endoscopic resection for local residual or recurrent cancer after definitive chemoradiotherapy or radiotherapy for esophageal squamous cell carcinoma', published in *Scientific Reports* 13(1): 10451 (2023), under Creative Commons Attribution 4.0.[111 112]

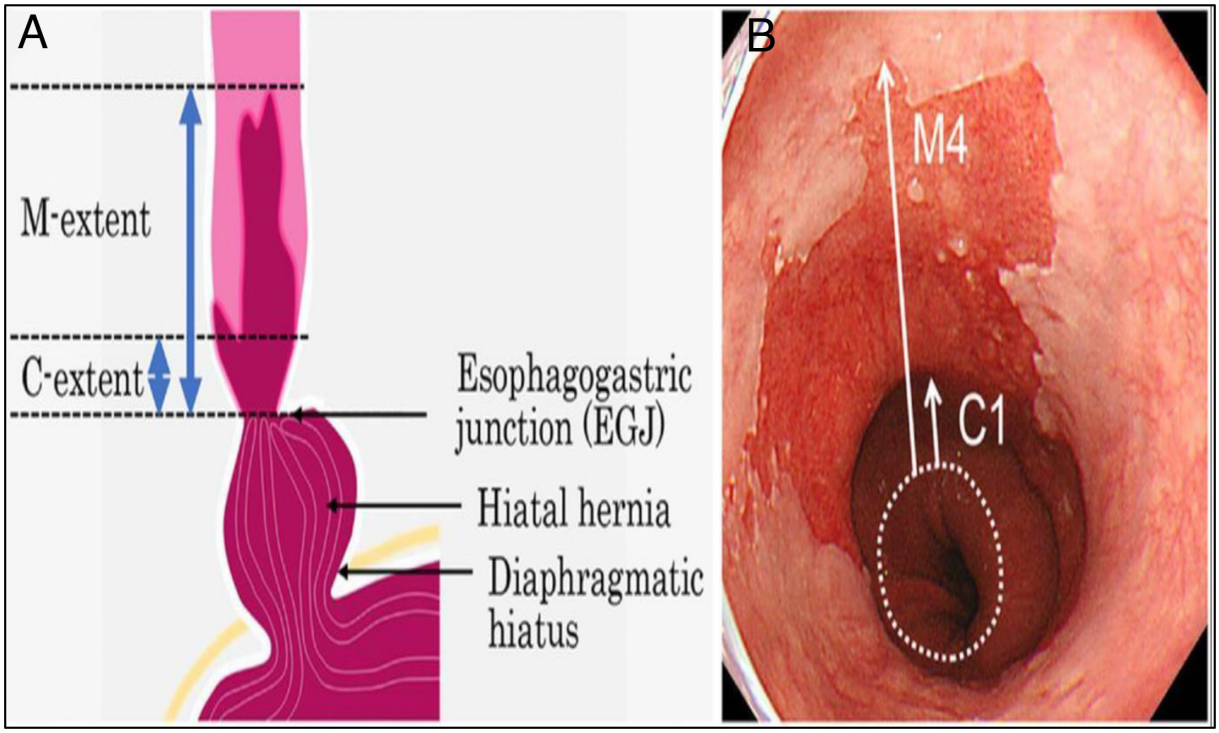

Figure 6 Prague C&M criteria. (A) Endoscopic criteria for a diagnosis of Barrett's oesophagus are described in the Prague classification. (B) In this case, the Prague classification is C1M4. Key steps. (1) Identify the gastro-oesophageal junction at the top of the gastric mucosal folds. If a hiatus hernia is present, note the level of the diaphragmatic pinch and the GOJ based on the level at the top of the gastric folds. (2) The distance from the GOJ to the top of the circumferential columnar mucosa is the C value in centimetres. (3) The distance from GOJ to the maximal extent of the columnar mucosa is the M-value Adapted from Ishimura N *et al*, 'Endoscopic diagnosis and screening of Barrett's esophagus: Inconsistency of diagnostic criteria between Japan and Western countries', published in *DEN Open* 2021 Nov 15;2(1):e73, under Creative Commons licence 4.0 [50,132]. GOJ, gastro-oesophageal junction.

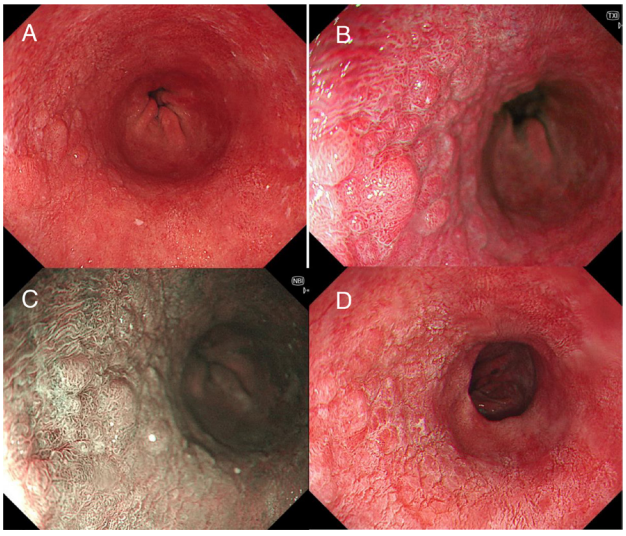

Figure 7 Barrett's high-grade dysplasia. (A) White light, (B) near focus, (C) NBI, (D) acetic acid. Reproduced with permission from National Endoscopy Training Programme, NHS Scotland Academy. NBI, narrow band imaging; NHS, National Health Service.

Key points

- Requires accurate assessment and reporting of the GOJ (distal end of the palisade vessels or proximal end of the gastric folds (figure 1).[46–51]
- Use the Prague Classification to describe the extent of the Barrett's segment (figure 6).[47 51 52] The length of a Barrett's segment is measured from the GOJ to the most proximal circumferential extent.[52] This is the 'C' value in the Prague classification. The maximum length of the most proximal extent of columnar mucosa from the GOJ, including tongues of Barrett's, is the 'M' value of the Prague classification. The M value should always be equal to or greater than the C value.
- Inspect using white light for a mucosal lesion.[47 51 53]
- Inspect using virtual chromoendoscopy for dysplasia.[22 23 51 52] Dysplastic areas are more vascular and will appear darker (figure 7).
- Applying acetic acid will increase lesion detection. Acetic acid should be applied using a spray catheter. Once applied, wait at least 1 min.[54] Neoplastic or high-grade dysplastic areas turn pale initially (acetowhite) and then turn red within 1 min (loss of acetowhite, figure 8).[55]
- Seattle protocol biopsies are still required to detect low grade dysplasia, given the poor sensitivity and specificity of enhanced imaging for this and the significantly increased risk of OAC in patients with low grade dysplasia complicating Barrett's oesophagus: four quadrant biopsies every 2 cm in addition to targeted biopsies of macroscopically visible lesions,[47 48 52 56] we uggest targeting 12, 3, 6 and 9 on the clock face.
- 12-3 O'clock is the area where Barrett's neoplasia is most commonly found.[57]

Surveillance

- If it is not possible to perform all biopsies at the endoscopy that diagnoses Barrett's oesophagus, an early repeat procedure should be performed within 6 months, provided there is no visible lesion in the Barrett's segment or dysplasia on biopsy.[38 58]
- 1 min inspection time per cm of Barrett's.[4 52 59 60]
- Photo-documentation of the landmarks, one image each cm of Barrett's, the GOJ in retroflexion and any visible lesion in white light, virtual chromoendoscopy and acetic acid.[4 52]
- Use the Prague classification.[4 47 51 52]
- Use the Paris classification for any visible lesion (figure 9).[4 47 52]
- Biopsies from all visible abnormalities/lesions/areas of nodularity or contact bleeding, followed by Seattle protocol—random four-quadrant biopsies every 2 cm of Barrett's oesophagus length.[4 47 51–53]

Surveillance Intervals for Barrett's oesophagus[52]

- <1 cm or irregular Z line no routine biopsies or surveillance.
- ≥1 cm–<3 cm every 5 years.
- ≥3 cm every 3 years.[47 52]

Consider stopping surveillance at 75 and/or <5 years life expectancy following discussion with the patient.

More scheduled endoscopy time is recommended for surveillance procedures depending on segment length and there is evidence that endoscopy lists dedicated to surveillance procedures by specialised endoscopists increase detection of dysplasia and cancer.[61]

REFLUX OESOPHAGITIS

Reflux oesophagitis is inflammation of the oesophageal mucosa secondary to gastro-oesophageal reflux disease (GORD), a condition in which

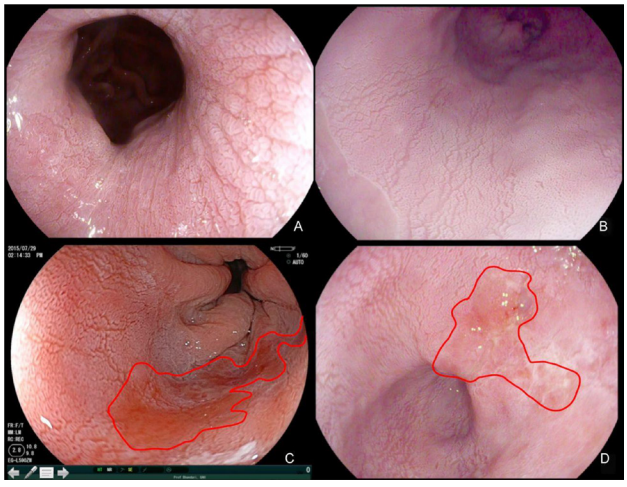

Figure 8 Examples of the acetowhitening effect on Barrett's mucosa. (A and B) Absence of focal loss of acetowhitening thereby indicating non-neoplastic Barrett's. (C) Presence of focal loss of acetowhitening highlighting an area of high-grade dysplasia. (D) Presence of focal loss of acetowhitening highlighting an area of intramucosal adenocarcinoma. Reproduced from *Gut*, Kandiah K., Chedgy FJQ, Subramaniam S, Longcroft-Wheaton G, Bassett P, Repici A, Sharma P, Pech O, Bhandari P, Repici A, Sharma P, Pech O, Bhandari P, 67, 2085–2091, 2018 with permission from *BMJ* Publishing Group licence number 596594089063769.

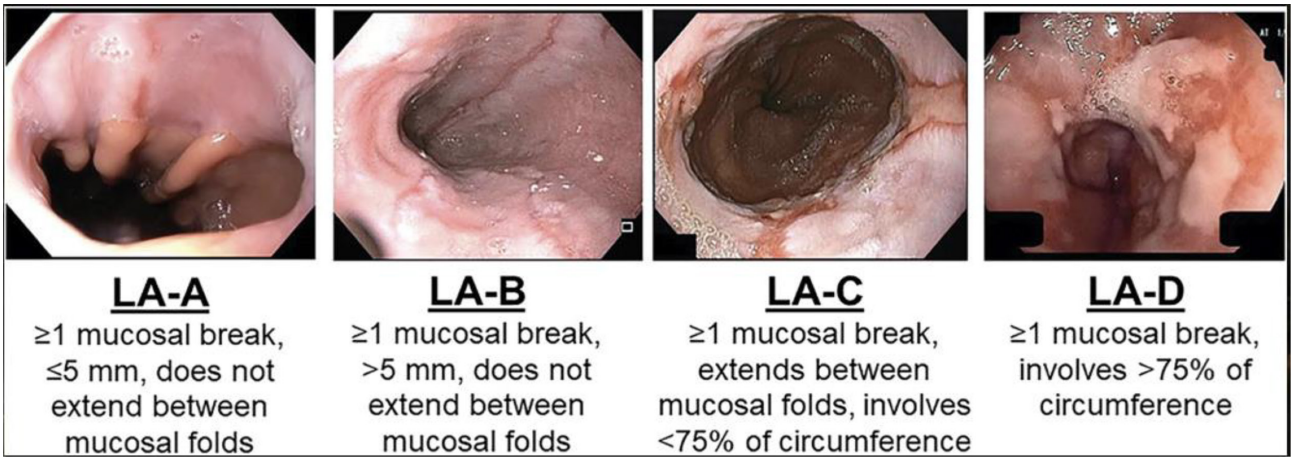

Figure 10 LA classification. Reproduced from Spechler S *et al*, 'Comparison of Los Angeles grades of erosive esophagitis scored by local investigators vs. central adjudicators in a clinical trial', published in *Clinical Gastroenterology and Hepatology*, under Creative Commons License 4.0, copyright Elsevier.[112 113] LA, Los Angeles.

the stomach contents reflux into the oesophagus causing troublesome symptoms and complications.[62] In Western countries, the prevalence of the disease is approximately 10% to 20%.[62] Less than 50% of patients with typical GORD symptoms have endoscopically recognisable mucosal lesions.[63] Due to its ease of use and lower interindividual variability in assessment, the Los Angeles (LA) classification of reflux oesophagitis should be used.[4] It is also recommended that other possibly related findings during endoscopy (eg, oesophageal stenosis or ulcer, Schatzki ring, Barrett's oesophagus) and the presence of hiatus hernia[2] should be documented.

Key points

- Patients with severe reflux oesophagitis (Los Angeles C/D, figure 10) should receive 6 weeks of PPI at double the standard dosage and follow-up endoscopy at 6 weeks if appropriate to patients given age and comorbidity, to check for healing and complications (eg, cancer or Barrett's).[2 38 64 65]
- Targeted biopsies from oesophagitis which is LA grade C or D or atypical in appearance, focusing on areas of mucosal abnormality, should be taken.[2 38 66 67]
- Particular attention should be focused above the GOJ at the 12–3 o'clock position and additional areas of nodularity or contact bleeding, as these are associated with dysplasia and cancer.[38 57]
- Patients with grade C/D oesophagitis almost always relapse on cessation of treatment and require long-term PPI treatment.

EOSINOPHILIC OESOPHAGITIS

Eosinophilic oesophagitis (EoE) is one of the most prevalent oesophageal diseases.[5] In adults, food bolus obstruction and dysphagia are strongly associated with a diagnosis of EoE.

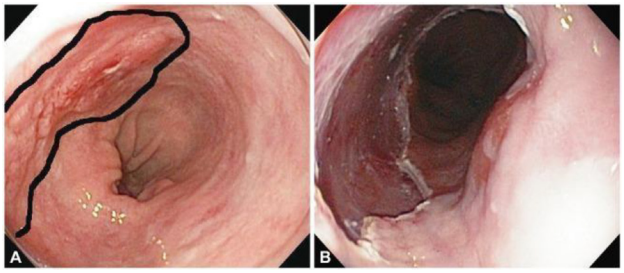

Figure 9 (A) Nodular lesion (0-IIa+IIb) 1 cm above GOJ in a Barrett's segment. The reported histology was intramucosal adenocarcinoma involving the muscularis mucosa—M3. (B) Mucosal defect after multiband resection. Reproduced from Aranda-Hernandez J *et al*, 'Treatment of dysplasia in Barrett's oesophagus', published in *Clinical Endoscopy* under Creative Commons Licence 3.0.[62 63] Reprinted with permission from the Korean Society of Gastrointestinal Endoscopy (Clin Endosc. 2014;47(1):55-64.). GOJ, gastro-oesophageal junction.

Key points

Diagnosis

1. Consider this diagnosis in all patients with dysphagia and food bolus obstruction.
2. If food bolus, then biopsy at time of removal. If spontaneous passage, then arrange outpatient endoscopy and biopsy and follow-up.[5]
3. Consider in all patients with dysphagia, even if the oesophagus is macroscopically normal.[5 68]

Endoscopic findings

- Oedema, rings, exudates, furrows and strictures,[68 69] fragile mucosa, tram-track erosions (figure 11).

Biopsy protocol and histopathology

1. Biopsies at least six from two different sites[2 38 68 70]: distal and proximal in separate pots or separate wells in a labelled cassette.
2. Diagnosis >15 eosinophils/ 0.3 mm^2 plus possibly other histological features (basal cell hyperplasia, oedema, eosinophil microabscesses, eosinophil layering, eosinophil degranulation, subepithelial sclerosis).[5]

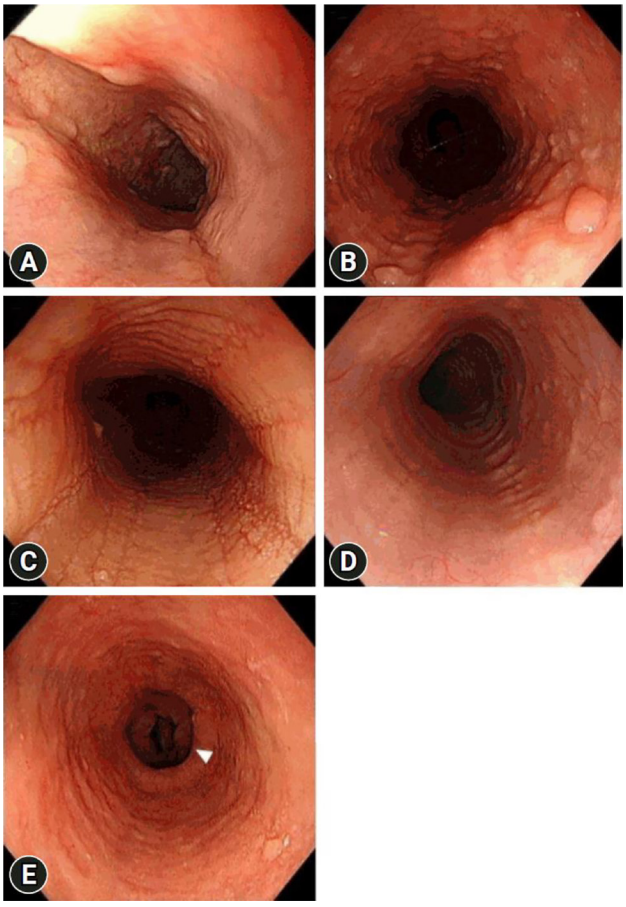

Figure 11 Endoscopic view of eosinophilic oesophagitis. (A) Mucosal oedema, (B) exudates, (C) furrows, (D) rings, (E) stricture (arrowhead). Reproduced from Yang E *et al*, 'Role of endoscopy in eosinophilic esophagitis', published in *Clinical Endoscopy*, under Creative Commons Attribution Non-Commercial Licence 4.0.[109 114] Reprinted with permission from the Korean Society of Gastrointestinal Endoscopy (Clin Endosc. 2025;58(1):1-9).

3. PPIs can interfere with histological diagnosis by lowering eosinophil numbers—consider repeat endoscopy after at least 3 weeks off PPI, *if still strong suspicion* of EoE after negative biopsy.[5]

Strictures

1. Occur in about 17%.[71] When found, biopsies should be taken[38] followed by commencement of topical steroids.[5] EoE strictures are safe to dilate and have a low risk of perforation (0.38%)[72] but mucosal tears are more common (9%).[73]

BSG guidelines on EoE can be found at: DOI: 10.1136/gutjnl-2022-327326

BSG guidelines on endoscopic dilation can be found at: DOI: 10.1136/gutjnl-2017-315414

OESOPHAGEAL VARICES

Oesophageal varices are dilated collateral blood vessels and a complication of portal hypertension, usually in the setting of cirrhosis.

Key points

There are a number of grading systems for oesophageal varices. The following is recommended[74];

- Grade 1: small straight oesophageal varices (<5 mm).
- Grade 2: enlarged (medium) tortuous varices occupying less than one third of the lumen.
- Grade 3: large coil-shaped varices occupying more than one third of the lumen (>5 mm).
- These correlate with the Japanese Research Society for Portal Hypertension grading system, F1, F2 and F3, respectively.

High-risk signs

- Red wale signs. These are red patches on varices, due to dilated intraepithelial veins under tension, indicative of recent or impending bleeding.
- White-nipple: This is a platelet-fibrin plug which indicates a site that recently bled.
- Cherry red spot: red spot suggestive of recent or impending bleeding.
- Haematocystic spots: crimson-coloured, large, blood-filled projections seen on the mucosal surface of a varix.

In addition to treating the cause of the portal hypertension, the management of oesophageal varices is pharmacological and endoscopic. Further information can be found in BSG Best Practice Guidance: outpatient management of cirrhosis—part 1: compensated cirrhosis DOI: 10.1136/flgastro-2023-102430[75]

HIATUS HERNIA

Hiatus hernia is overdiagnosed and overattributed with patient symptoms. There are two systems for describing the size of a hiatus hernia. The first is the axial length between the diaphragmatic crura and the GOJ. A distance of ≥2 cm is required for the diagnosis of hiatus hernia.[2] The other system is the Hill Classification (figure 12). It is important to retroflex into a hiatus hernia to adequately view the cardia and GOJ. Large hiatal hernias can be associated with mucosal ulcerations from ischaemia from friction between the gastric wall and diaphragmatic hiatus. Such a Cameron ulcer can result in chronic iron deficiency anaemia.

Key points

- There are 3 types of hiatus hernia:

Type 1: Sliding (axial). The GOJ and sometimes the cardia 'slide' in a supradiaphragmatic direction. This accounts for greater than 95% of hiatus hernias.[76]

Type 2: Paraoesophageal. The GOJ or cardia herniate above the diaphragm and come to lie alongside the oesophagus.

Type 3: Mix of types 1 and 2.

There are two systems for describing the size of a hiatus hernia. The first is the axial length between the diaphragmatic pinch and the GOJ. A distance of ≥2 cm is required for the diagnosis of hiatus hernia.[2]

- Hill Classification of the gastro-oesophageal flap valve (GEFV).

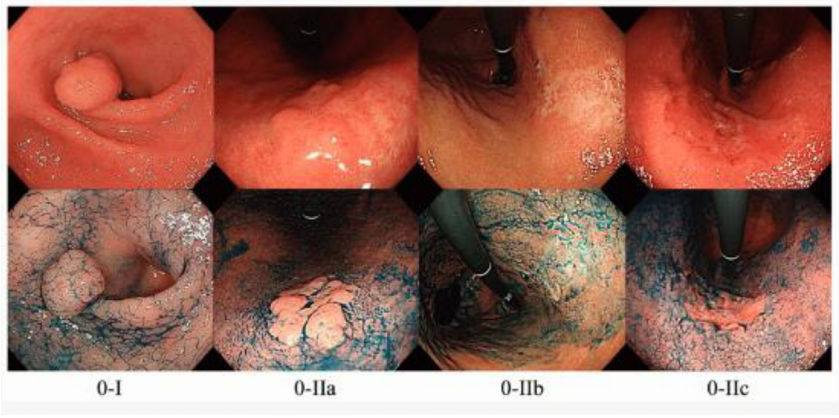

Figure 13 Paris classification (upper row: white light only; lower row: same lesions under indigo carmine chromoendoscopy). (A) Mass, Paris 0-I, (B) unclassified, Paris 0-IIa, (C) ulcerative, Paris 0-IIb, (D) ulcerative, Paris 0-IIc. Reproduced from Fujiuoshi R, 'Endoscopic classifications of early gastric cancer: A literature review', published by *Cancers (Basel)* 2021 Dec 26;14(1):100, under Creative Commons Attribution License 4.0.[112 115]

This provides a grading system for the competence of the GOJ. A higher Hill grade is associated with reduced lower oesophageal sphincter pressures and need for/response to PPI. The Hill grades are[77]:

Grade 1: prominent fold in the cardia along lesser curve (GEFV) closely opposed to endoscope.

Grade 2: GEFV present, but slightly less defined and rarely opens with respiration and closes promptly.

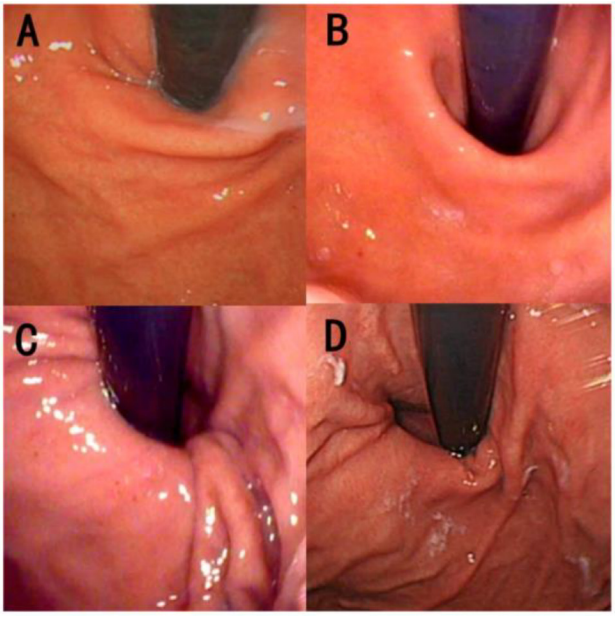

Figure 12 Hill's classification. (A) Grade I: a prominent fold of tissue along the lesser curvature and closely apposed to the endoscope. (B) Grade II: the fold is present, but there are periods of opening and rapid closing around the scope. (C) Grade III: the ridge is barely present, and there is often failure to close around the scope. (D) Grade IV: the muscular ridge is absent, and the gastroesophageal area continuously remains open. A hiatal hernia is always present. Reproduced from Wu W *et al*, 'Reflux finding score is associated with gastroesophageal flap valve status in patients with laryngopharyngeal reflux disease: a retrospective study.', published in *Scientific Reports* 2019 Oct 31;9(1):15744, under Creative Commons Attribution 4.0 International Licence [51,139].

Grade 3: GEFV is barely present, failure to close around scope. It is nearly always accompanied by a hiatus hernia.

Grade 4: GEFV is absent, GOJ is continuously open and squamous epithelium can often be seen from the retroflexed position. Hiatal hernia is always present.

Indications for treatment

Paraoesophageal and mixed hiatus hernia can present with pain, incarceration and chronic bleeding from Cameron ulcers. These may be an indication for surgery.

For endoscopic pictures following fundoplication as well as complications post fundoplication, please visit this article doi: 10.21037/ales-20-48.

GASTRIC CANCER

A recent English study of 106 557 patients found that 8.5% of them had undergone OGD in the preceding 3 years that did not diagnose cancer. 36% had potentially missed gastric cancers.[39] Focal erythema or whitish discolouration, irregular mucosal surface with protrusions, elevations or depressions, spontaneous bleeding and abnormal mucosal folds are the most important hallmarks of early gastric cancer.[78]

Key points

Basic classification of gastric cancer

- Gross tumour morphology is categorised as either superficial or advanced.[39]
- Superficial tumours should also be characterised by the Paris classification[79] as polypoid (type I), flat (type II) or excavated (type III) lesions. Flat type II lesions might also have some elevation which is less than 1.3 mm (IIa), or they might be completely flat (IIb) or superficially depressed (IIc) (figures 13 and 14). Flat, depressed or excavated lesions have a significantly higher chance of submucosal invasion, which influences their endoscopic resectability.[78]

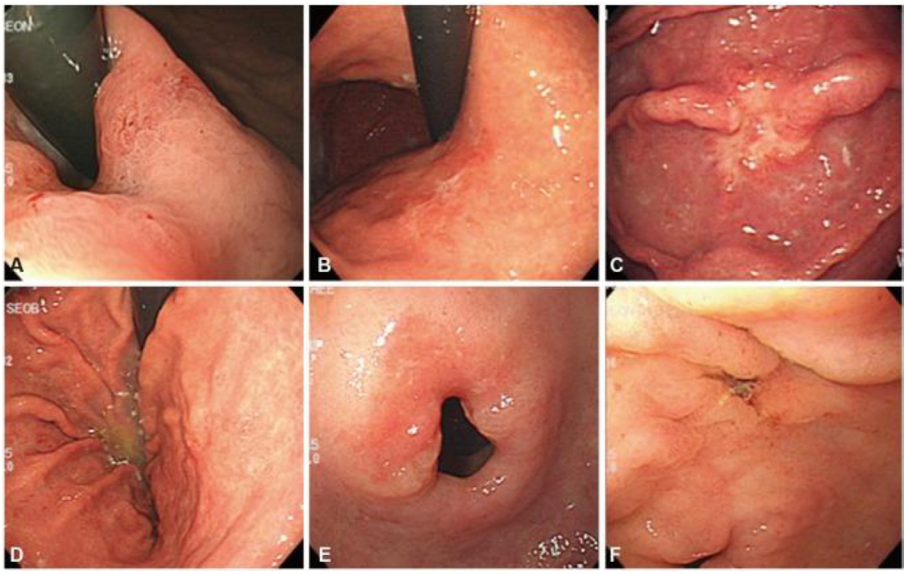

Figure 14 Endoscopic findings of various EGC lesions in blind spot areas. (A) A flat erythematous lesion (EGC 0-IIb) at the cardia. (B) An irregular flat lesion (EGC 0-IIb) in the posterior wall of the upper body. (C) A disrupted mucosal fold (EGC 0-IIc) in the greater curvature of the upper body. (D) A discoloured flat lesion (EGC 0-IIb) in the posterior wall of the lower body. (E) A reddish flat lesion (EGC 0-IIb) in the P-ring. (F) A well-demarcated depressed lesion (EGC 0-IIc) in the lesser curvature of the antrum. Reproduced from Moon H *et al*, 'Improving the endoscopic detection rate in patients with early gastric cancer', published by *Clinical Endoscopy*, under Creative Commons Attribution Non-commercial Licence 3.0.[116 117] Reprinted with permission from the Korean Society of Gastrointestinal Endoscopy (Clin Endosc. 2015;48(4):291-296). EGC, early gastric cancer.

- When detected, describe in terms of morphology, location and size. The tumour location can be: upper, middle or lower third of the stomach or involving the GOJ. A GOJ tumour's location extends to 2 cm above to 2 cm below the GOJ. Furthermore, it is important to describe the location of the tumour along the cross-sectional aspect of the stomach (anterior, posterior, lesser or greater curve).
- Advanced gastric cancers should have at least eight biopsies taken but only 1–2 from potential early cancers to avoid compromising potential later endoscopic resection.[38] In cases of suspected linitis plastica, at least ten bite-on-bite biopsies should be taken from areas of mucosal abnormality or poor distensibility.[38]
- Online learning to help identify early gastric neoplasia: Higan project https://www.higan-npo.com/e-learning-endoscopy

GASTRIC ATROPHY AND INTESTINAL METAPLASIA

Chronic atrophic gastritis and gastric intestinal metaplasia are caused primarily by chronic *H. pylori* infection and less commonly by autoimmune gastritis. The 5-year incidence of gastric cancer is approximately 10% in patients with extensive intestinal metaplasia or gastric atrophy.[29] The aim of endoscopic surveillance of high-risk patients is to enable early detection of gastric adenocarcinoma at a stage where curative treatment is possible (figure 15).

Recent data from a multicentre study in the UK showed that 75% of gastric atrophy/intestinal metaplasia detected on biopsy was missed by endoscopists during endoscopy.[80]

Key points

- The primary endoscopic trigger to diagnosis is loss of the gastric folds in the body and mucosal pallor with visible

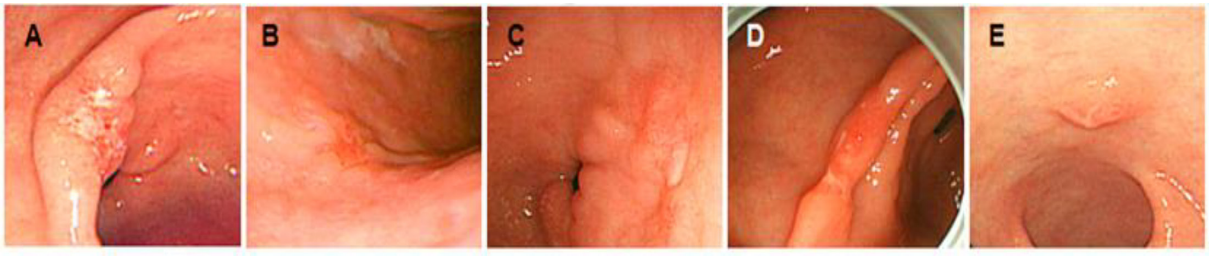

Figure 15 (A–E) Examples of early gastric cancers. Adapted from Kang HS *et al*, 'Molecular risk markers related to local tumor recurrence at histological margin-free endoscopically resected early gastric cancers: A pilot study', published in *Pathology-Research and Practice* 2021 Jun;222:153434, under Creative Commons License 4.0.[112 118]

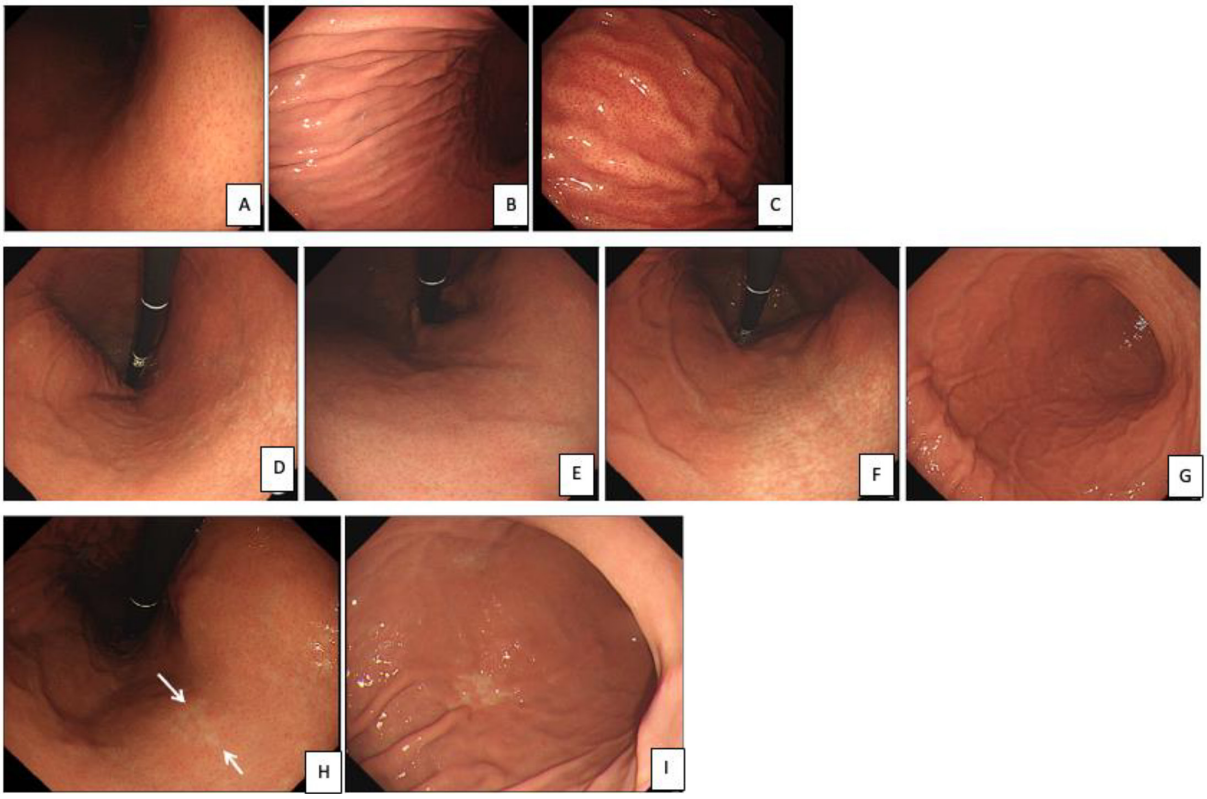

Figure 16 Top row: endoscopic images showing regular arrangement of collecting venules across the gastric body as follows: (A) Along the lesser curve, (B) along the greater curve, (C) in the high gastric body along the greater curve. Middle row: gastric atrophy. (D) Closed type 2 with atrophic changes involving the low gastric body along the lesser curve. (E) Closed type 2 with normal regular arrangement of collecting venules on the lesser curve aspect of the high gastric body. (F) Closed type 3 with atrophic changes involving the gastric body along the lesser curvature. (G) Closed type 3 with normal regular arrangement of collecting venules in the anterior and posterior walls of the gastric body. Bottom row: (H, arrow and I) endoscopic findings showing a focal lesion in the midgastric body along the lesser curvature and a discoloured lesion involving the low gastric body along the greater curvature. (I) The biopsy showed diffuse type carcinoma at both sites. Adapted from Oh J-H *et al*, 'Endoscopic findings of common gastritis in Koreans', published in *The Korean Journal of Helicobacter and Upper Gastrointestinal Research* 2023;23(2):99-107, under Creative Commons Attribution Non-commercial Licence 4.0.[109 119]

vessels due to atrophy[81] (figure 16) or pale patches of intestinal metaplasia usually in the antrum (figure 17).

- If suspected, close inspection on white light for a discrete lesion and virtual chromoendoscopy[29 82 83] to identify dysplasia within areas of atrophy or intestinal metaplasia should be performed.
- Biopsies should be taken for Helicobacter pylori or a stool antigen test completed.[84]
- Diagnosis requires Sydney protocol biopsies (see below) and biopsies of any discrete lesion.[84 85]
- Surveillance at 3 years is only offered if the patient is likely to benefit from this (they would potentially be fit for endoscopic intervention in three years' time) and there is extensive atrophy or intestinal metaplasia i.e. involving the gastric body. Surveillance should also be considered if there is antral atrophy or intestinal metaplasia only and:
  - There is a strong family history of gastric cancer.[65 85]
  - Persistent *Helicobacter* infection.[29 65 85]
- During surveillance, further biopsies are not needed unless image enhancement raises suspicion of a neoplastic lesion.[83]
- If an early neoplastic lesion is found, two biopsies should be taken.

Link to BSG guidelines on patients at risk of gastric adenocarcinoma: DOI: 10.1136/gutjnl-2018-318126

In patients with iron deficiency anaemia, gastric biopsy specimens for *H. pylori* and atrophic gastritis are not recommended in the absence of endoscopic findings suggestive of atrophy.[86 87]

Biopsies for diagnosis (Sydney biopsy protocol)[4 84]

- Two from the antrum at least 2 cm from pylorus along the lesser and greater curve if diffuse mucosal changes or from endoscopically suspected atrophy or intestinal metaplasia if focal in one pot or separate compartment within a cassette.
- One from the incisura in the same antral biopsy pot or compartment within a cassette.
- Two from the gastric corpus (one from lesser curve, another from greater curve) if diffuse mucosal changes or from endoscopically suspected atrophy or intestinal metaplasia if focal in a second pot or separate compartment within a cassette.[29 38 88]

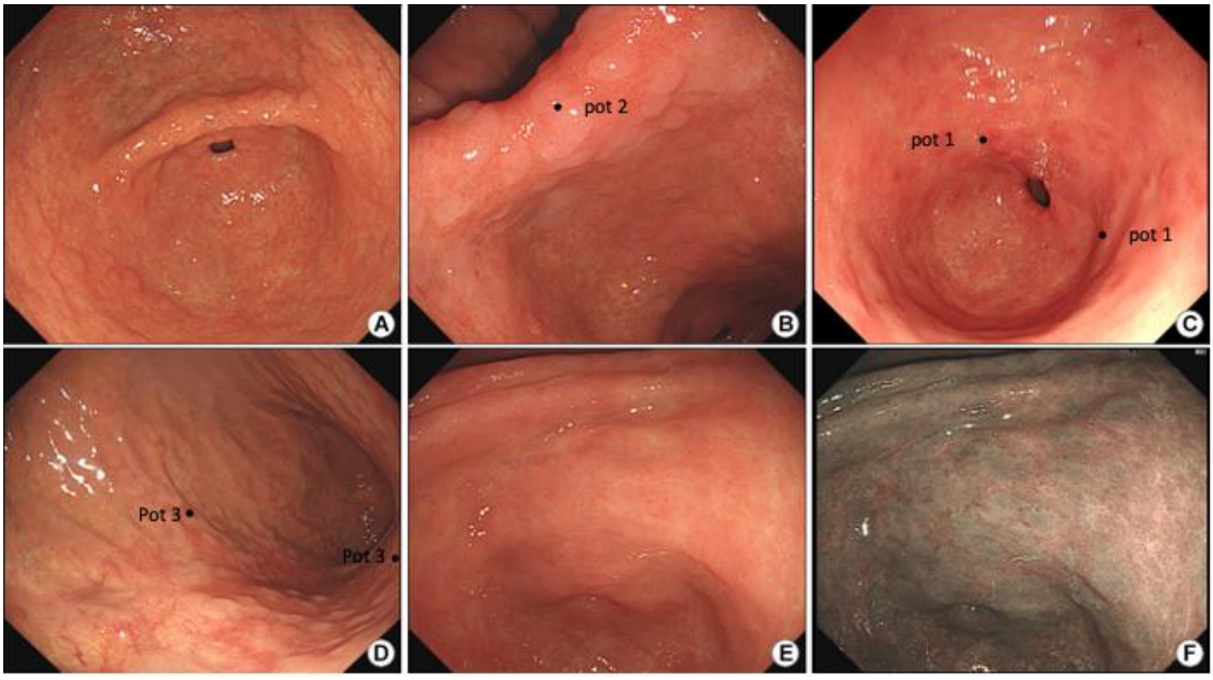

Figure 17 Endoscopic findings showing intestinal metaplasia. (A, B) Elevated white plaques (whitish granular plaques) are observed in the antrum (A) and at the gastric angle (B). (C, D) Elevated plaques are surrounded by a mixed patchy erythematous mucosal area in the antrum (C) and gastric body (D). (E, F) Grey plaques are visualised in the antrum (E); the plaques are more prominently observed using NBI (F). Sydney protocol is integrated into the images and should be labelled as 'random' or 'targeted'. Pots 1–3 indicate Sydney protocol biopsy sites. Adapted from Oh J-H *et al*, 'Endoscopic findings of common gastritis in Koreans', published in *The Korean Journal of Helicobacter and Upper Gastrointestinal Research* 2023;23(2):99-107, under Creative Commons Attribution Non-commercial Licence 4.0.[92 109] NBI, narrow band imaging.

Target the biopsies to the areas of highest risk of atrophy or metaplasia from virtual chromoendoscopy.[85] Targeted biopsies should be obtained from any visible mucosal abnormalities and placed in appropriately labelled specimen pots.[84] Biopsy samples should be labelled either as 'directed' or 'random'.[29]

GASTRIC ULCER ASSESSMENT AND FOLLOW-UP

Gastric ulcer is defined as a mucosal defect >5 mm[2 89] that penetrates through to the muscularis mucosa and submucosa. Gastric ulcers usually result from *H. pylori* or non-steroidal anti-inflammatory drug use.

Key points

- Ulcer size and location should be described.
- Ulcers should be photographed on white light and virtual chromoendoscopy.
- Testing for *H. pylori* is recommended for gastric ulcers with a rapid urease test and on biopsies of the ulcer.[2] If the urease test and histology for *H. pylori* are negative in a patient already on a PPI or who has had antibiotics in the past month, consider performing a stool antigen test post procedure, ensuring the patient has been off PPI for 2 weeks. *Helicobacter* eradication should be provided if there is a positive urease or stool antigen test.[2]
- In the case of treatment-resistant *H. pylori*, biopsies for *H. pylori* culture and sensitivity need to be taken before any contact of the biopsy forceps with fixation fluid for other biopsies for histology. The biopsies need to be placed in a sterile universal container with a small amount of isotonic saline.[90] Biopsies should be taken from both the antrum and corpus.[91 92]
- If ulcer biopsies are not taken (eg, due to the risk of bleeding), repeat endoscopy within 2 weeks is advised.[38]
- The BSG and JAG recommend a repeat endoscopy within 12 weeks of diagnosis to assess healing and consider further biopsies if appropriate to the patient depending on age and comorbidity.[2 93]

Macroscopic assessment

1. Potential malignant characteristic of ulcers: larger solitary, non-antral, discoloured base and elevated or irregular border. The base is considered discoloured if areas with necrosis and irregularity of different colours are present. The border is considered elevated in proportion to the centre of the base and the mucosa surrounding the ulcer. An irregular border means asymmetrical.[94] Other signs of potentially malignant ulcers are raised edges with contact bleeding and gastric fold disruption. Non-malignant ulcers tend to occur in the antrum, are smaller with normal surrounding mucosa and have well-defined margins.[9495]
2. Assessment using white light, near focus or magnification and virtual chromoendoscopy is recommended.
3. Photographs should be taken on white light and virtual chromoendoscopy.

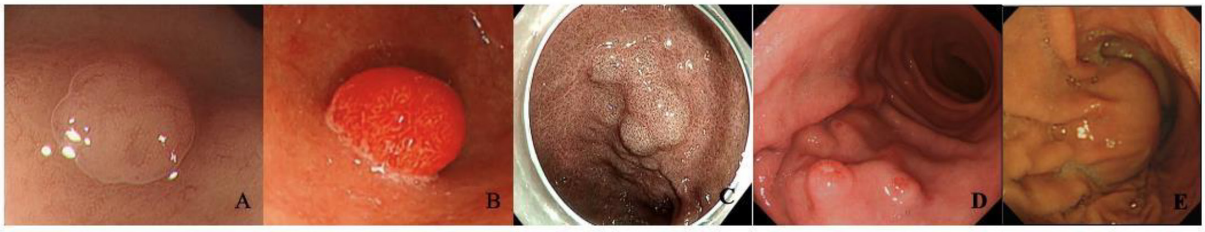

Figure 18 Gastric polyps. (A) Fundic gland polyp, (B) hyperplastic polyps, (C) gastric adenoma, (D) neuroendocrine tumour, (E) submucosal lesion Images reproduced with permission from National Endoscopy Training Academy, NHS Scotland. NHS, National Health Service.

4. At least six targeted biopsies of the edge and base are recommended.[11 38 70] If there are potentially malignant features, eight biopsies are recommended, targeting areas of potential dysplasia (eg, nodular or irregular areas). Biopsies of the ulcer base are recommended if the base is irregular or partially healed, as this has been shown to increase the pick-up rate of cancer.[38 94 96]

In a recent study, 778 patients with ulcers were identified and 60.3% had a follow-up endoscopy. 8.6% (66/778) were diagnosed with cancer. No cases of cancer were found on follow-up endoscopy of a benign appearing ulcer with negative biopsies.[95] This study proposes risk stratification for all ulcers found at index endoscopy depending on:

- Size: *0*—<1.25 cm, *2*—1.25 cm–2.99 cm, *3*—>3.00 cm.
- Site: antral *0*, non-antral *1*.
- Age: *0*—<68, *1*—68–79, *2*—>80.

The higher the score, the higher the risk of malignancy.

GASTRIC POLYPS

>90% are asymptomatic. Rarely, symptoms may occur due to bleeding. When identified, polyps should be described using the Paris classification, number, size and location and photographed.

Targeted biopsies for hyperplastic, adenomatous and neuroendocrine polyps are recommended (figure 18).[38] Background mucosa should be assessed for chronic atrophic gastritis and Sydney protocol performed if atrophy is present in the context of a hyperplastic or adenomatous polyp.[38] Fundic gland polyps should only be biopsied if in the antrum, >1 cm, ulcerated or atypical in appearance.[38]

DUODENAL ADENOMAS

Duodenal adenomas are subdivided into ampullary and non-ampullary.[97] They occur in 0.03%–0.4% of upper GI endoscopies. Duodenal adenomas are less often sporadic than gastric or colonic adenomas and 60% are associated with familial adenomatous polyposis.[98] For endoscopic surveillance of FAP and patients with a first degree relative with a FAP diagnosis, please refer to BSG family risk guidance DOI: 10.1136/gutjnl-2019-319915. The other predisposing genetic syndromes are MUTYH-associated polyposis syndromes.[99] Sporadic adenomas are associated with smoking, Barrett's oesophagus, fundic gland and colorectal polyps.[97] Sporadic adenoma incidence increases with age. Ampullary adenomas are more often sporadic, but up to 50% of ampullary adenomas will harbour a focus of adenocarcinoma.[100]

The European Society of Gastrointestinal Endoscopy (ESGE) guidance[97] is to remove all adenomas. However, duodenal adenoma management requires complex clinical decision making due to patient comorbidity, high complication rates and recurrence rates following endoscopic resection.

Key points

Therefore, the following is recommended for all adenomas:

- Assessment of,
  - Size.
  - Morphology using the Paris classification
  - Pit pattern.
- Photographs of the entire lesion using white light and virtual chromoendoscopy.

Cap-assisted endoscopy and near focus can be helpful for assessment and photo-documentation.

- A side viewing endoscope where there is extension into the major or minor papilla.[97]
- A routine biopsy is not recommended except in the presence of endoscopically diagnosed malignancy (more common in lesions ≥2 cm, Paris 2c, demarcation line within polyp, amorphous pit pattern).
- ESGE suggests that if endoscopic features are suggestive of superficial duodenal adenoma, the use of biopsy for histological assessment should be limited prior to endoscopic resection, since its additional diagnostic yield might be limited and resection might be compromised.[97]
- If there is a high confidence optical diagnosis of a duodenal adenoma, then patients can be referred directly for endoscopic mucosal resection with appropriate images without biopsy.[38]
- For ampullary adenomas, Endoscopic Ultrasound is also recommended if intervention is clinically appropriate.

The risk of duodenal adenocarcinoma in familial polyposis can be graded using the Spigelman classification. The higher the score, the higher the risk. Following assessment and documentation,

discussion with or referral to clinicians with expertise in their management is recommended. Where a patient is found to have a duodenal adenoma, a screening colonoscopy should be considered.[101–104]

COELIAC DISEASE

Recent guidance indicates that patients with no alarm symptoms with tissue transglutaminase (TTG) ≥10 × upper limit of normal (ULN) do not require an endoscopy to confirm coeliac disease.[38 105] Analysis of the National Endoscopy Database has shown that in 1 year in the UK, 499 278 biopsies from 27.4% of patients undergoing diagnostic UGI endoscopy were taken from the duodenum. Endoscopy services should ensure coeliac serology is tested prior to endoscopy in all patients referred for weight loss or iron deficiency anaemia, given the high negative predictive value, cost saving and environmental benefit.[38]

Consider duodenal biopsies in the following circumstances[38]:

- Treatment-refractory iron deficiency anaemia with no other cause, irrespective of tTg level.
- Weight loss and symptoms suggestive of malabsorption with no other cause, irrespective of tTg level.
- Positive tTg but less than ×10 ULN or if there is clinical suspicion of coeliac disease but tTg not checked prior to endoscopy, to avoid repeating if tTg is later found to be raised but < ×10 ULN.

Where endoscopy and biopsies are indicated:

- Minimum biopsy set would be four biopsies from D2 and 2 from the duodenal bulb to account for the possibility of patchy distribution[38 106] on a gluten containing diet, that is, 2 weeks eating two slices of bread per day.[107] These samples should be placed in two separate containers or separate compartments in a labelled cassette. One biopsy per pass is recommended to improve the diagnostic yield.[108]
- Link to guidelines for best practices in monitoring established coeliac disease: DOI: 10.1038/s41575-023-00872-2.

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