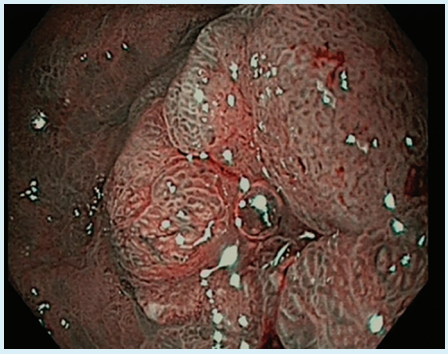
Mistakes in endoscopy and how to avoid them

Arnaud Lemmers and Jacques Devière



Upper and lower gastrointestinal endoscopy examinations are performed daily as routine diagnostic procedures in a large number of patients with nonspecific indications, such as heartburn, pain, anaemia, bleeding, workup of portal hypertension and so on. Most of the examinations will point to a classic diagnosis (e.g. peptic disease, cancer, variceal management), but sometimes we see patients who've had multiple diagnostic endoscopic procedures in the previous few months with nonconclusive findings. The diagnostic mistakes discussed here are those that sprang to mind based on our endoscopic experience and they are discussed in an evidence-based approach. For therapeutic endoscopic procedures (e.g. ERCP and resections), we present the most important mistakes that are often seen in our practice and have major consequences for the patient. We propose, from our experience, a simple approach to avoid these mistakes.

Mistake 1 Missing a diagnosis of Cameron ulcers

Cameron ulcers were first described in 1986 by Cameron and Higgins.[1] These erosions, or ulcerations, in the gastric mucosa are located at the diaphragmatic hiatus and consist in multiple linear lesions on the crests of gastric folds. They are associated with upper gastrointestinal haemorrhage or obscure bleeding. Identifying Cameron ulcers requires antegrade and retrograde observation of the neck of the hiatal hernia and they often go unrecognized during upper endoscopy. The overall mean number of hospitalizations for upper gastrointestinal bleeding without any identified bleeding source preceding a final diagnosis of Cameron ulcers was 3.4 in a series of 16 patients published in 2013.[2] The incidence of Cameron ulcers as the source of severe upper GI bleeding was low (0.2%), but was higher as the source of obscure GI bleeding (3.8%). All patients had a large hiatal hernia (>5cm) and their mean age was 70 years old.[2]

So, for patients who have a large hiatal hernia and unexplained anaemia or upper GI bleeding, we recommend paying close attention to the cardia in both an antegrade and retrograde manner (figure 1).

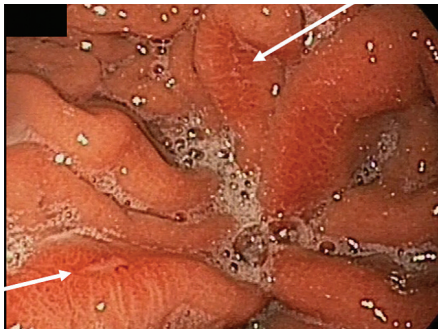


Figure 1 | Antegrade endoscopic view of the neck of a hiatal hernia in a 53-year-old man with iron deficiency anaemia of unknown origin diagnosed 6 years previously. The white arrows indicate the presence of Cameron ulcers. The patient was successfully treated with a PPI and iron supplementation.

Mistake 2 Missing a diagnosis of Dieulafoy lesions

Described by Gallarden in 1884 and Georges Dieulafoy in 1898,[3] the Dieulafoy lesion is an abnormal vessel of the mucosa. Normally, vessels become smaller as they penetrate the mucosa, but the Dieulafoy lesion is a calibre-persistent arteriole that remains abnormally large and protrudes through the normal mucosa into the lumen. A small mucosal defect with the eruption of the vessel can cause bleeding. Although they can be located elsewhere in the gastrointestinal tract, the most frequent location of a Dieulafoy lesion is the proximal stomach along the lesser curve.[4] Representing only 1–5% of upper gastrointestinal bleeding cases, the Dieulafoy lesion is often unrecognized and multiple gastroscopies may be needed for it to be identified (figure 2).[5]

The most frequent presentation of the Dieulafoy lesion is acute overt bleeding with haematemesis and/or melena. The success of endoscopic haemostasis done by a combined method (adrenaline injection and thermal electrocoagulation) or mechanical methods has been reported to reach 90%, with a higher endoscopic diagnostic and therapeutic yield when endoscopy is performed sooner.[5] The rebleeding rate has been reported to be as high as 9–40%.[6] In our experience, it happens that a Dieulafoy lesion is suspected in patients who experience multiples episodes of upper gastrointestinal

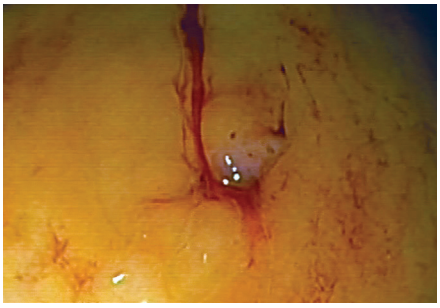


Figure 2 | Active bleeding from a Dieulafoy lesion in the proximal stomach of a 69-year-old patient. The patient presented with recurrent severe upper gastrointestinal bleeding, underwent more than 3 gastroscopies and was transfused with more than 8 red blood cell (RBC) units before the bleeding source was identified. The lesion was successfully clipped with no further bleeding recurrence.

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Arnaud Lemmers and Jacques Devière are at the Department of Gastroenterology, Hepatopancreatology and Digestive Oncology, Erasme Hospital, Université Libre de Bruxelles (ULB), Brussels, Belgium.

All images courtesy of: A. Lemmers and J. Devière.

Correspondence to:
arnaud.lemmers@erasme.ulb.ac.be
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bleeding without any origin visualized during multiples gastroscopies.

There are tips and tricks that can be followed to help find the Dieulafoy lesion. First, opt for an urgent upper endoscopy in the case of a new episode of bleeding or dizziness (before blood exteriorization). Second, ask the patient to cough when the endoscope is inside the stomach and no visible lesion is seen–this could increase the vascular pressure and trigger bleeding, leading to adequate endoscopic treatment.

Mistake 3 Missing a diagnosis of eosinophilic oesophagitis

Described for more than 15 years, eosinophilic oesophagitis (EoE) has become increasingly recognized. Although, in our routine clinical practice, many patients with EoE have already undergone multiples endoscopies before the diagnosis is established.

The major symptoms associated with EoE in adults are dysphagia and food impaction, but secondary symptoms of heartburn and atypical noncardiac chest pain have also been reported.[7] About 70% of patients with EoE have asthma, allergic rhinitis and/or atopic dermatitis, underlining the association with atopy. Endoscopic signs suggestive of EoE are: the presence of rings (trachealization), oedema (loss of vascular marking), exudates (white plates), furrows (vertical lines) and strictures (figure 3). The diagnosis is made by obtaining multiple biopsies (2–4) of the distal and proximal oesophagus, showing mucosal eosinophilia. Pathophysiologic explanations include gastro-oesophageal reflux disease (GERD) and food antigen sensitization or allergy. Proton pump inhibitor (PPI) therapy is the cornerstone of treatment and its role has been attributed to the direct anti-inflammatory properties of PPIs and the repair of mucosal permeability defects. If needed, further treatment is based on topical steroid treatment and the six food (milk, wheat, soy, egg, nuts and seafood) elimination diet.[7]

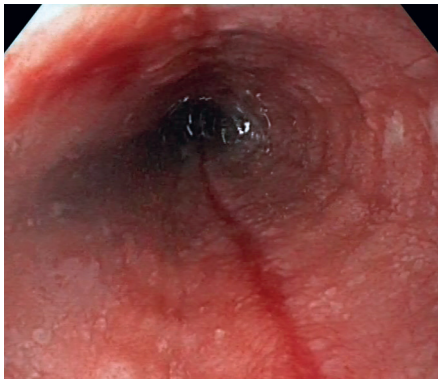

Figure 3 | Eosinophilic oesophagitis (EoE) in a 53-year-old man who presented with atypical recurrent acute episodes of retrosternal chest pain, atopy and peripheral hypereosinophilia. The gastroscopy revealed typical features of EoE–multiples exudates and furrows. PPIs and the six food elimination diet were prescribed to the patient and achieved a good clinical response.

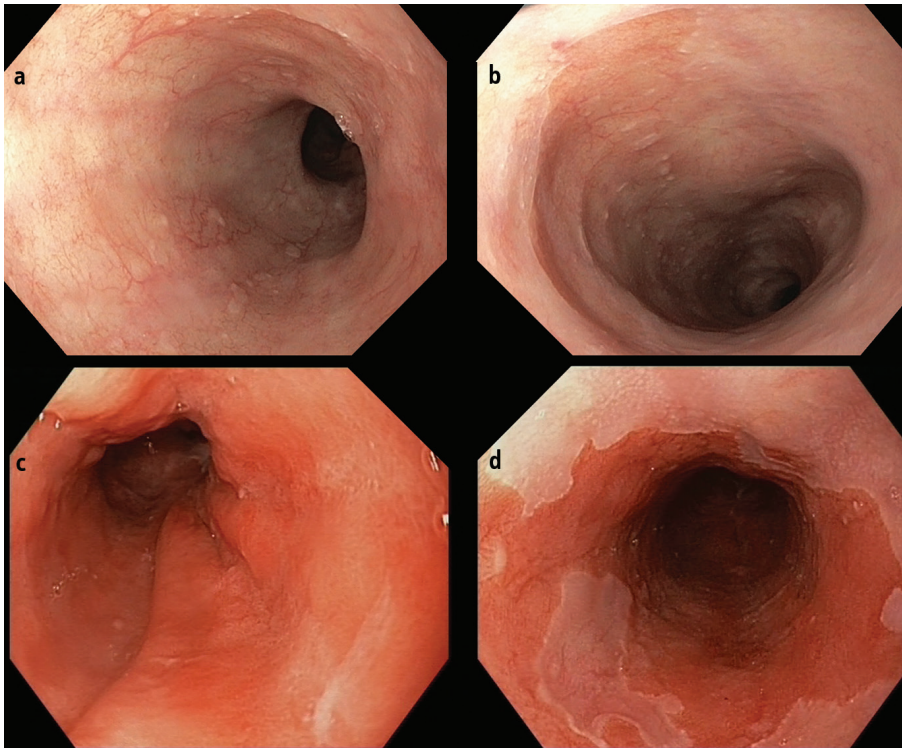

Figure 4 | Long-segment Barrett oesophagus. **a** and **b** | A C12M17 long Barrett oesophagus in a 73-year-old man. **a** | the Z line is difficult to identify from 11h to 2h in this photo taken at 28cm from the incisors. If no active search for the the Z line is done, the diagnosis might be missed. **b** | the scope is retrieved to 23cm to see the upper part of the Z line. **c** and **d** | a 72-year-old man presented with a C9M10 Barrett oesophagus. **c** | No Z line can be identified in the endoscopic view at 33cm from incisors and the diagnosis of Barrett oesophagus could be missed. d | At 30cm, the irregular Z line can clearly be identified.

Mistake 4 Missing a diagnosis of long-segment Barrett oesophagus

Barrett oesophagus is defined by the replacement of squamous epithelium by columnar epithelium, with intestinal metaplasia identifiable in biopsy samples taken from the distal oesophagus. On endoscopy, Barrett oesophagus appears as a salmon-pink tongue of mucosa extending into the oesophagus from the gastroesophageal junction.[8]

Long-segment Barrett oesophagus is associated with an increased risk of malignancy. Classically, Barrett oesophagus extension is defined by the Prague C and M criteria,[9] providing the maximal circumferential (C) and maximal tongue (M) extension in cm. The length of Barrett oesophagus is associated with malignancy risk and the number of biopsies needed to be taken for detection of dysplasia.

In case of very long Barrett oesophagus, in some patients the Z line (the junction of the columnar epithelium and the squamous epithelium) is so high in the proximal oesophagus that some endoscopists do not notice the presence of Barrett oesophagus. In those cases, if no active search is done to locate the Z line when handling the scope, it is likely that the identification of Barrett oesophagus will be missed, with unfortunate consequences for the patient in terms of dysplasia diagnosis and a surveillance plan (figure 4).

Mistake 5 Confusing the diagnosis of gastric antral vascular ectasia lesions, portal hypertensive polyps and portal hypertensive gastropathy

Confusion can occur in the diagnosis of gastric lesions associated with portal hypertension. In patients with portal

hypertension, various gastric lesions may occur that are known to present in different forms and require different management. In our experience, confusion between portal hypertensive gastropathy (PHG) and gastric antral vascular ectasia (GAVE) frequently occurs (figure 5). Sometimes, gastric polyps may also be associated with portal hypertension.

PHG classically starts from the fundus and corpus and extends into the antrum. By contrast, GAVE starts in the antrum and extends to the corpus. PHG has a snakeskin or mosaic background mucosa and, when severe, is associated with flat or bulging red or brown spots that may be friable or frank bleeding in severe cases. GAVE lesions are characterized by convoluted and tortuous columns of ectatic vessels along each longitudinal fold of the antrum, converging at the pylorus, which look like the stripes of a watermelon. Biopsy samples, if needed, can help the diagnosis (PHG is associated with dilated mucosal and submucosal veins; GAVE is associated with dilated mucosal capillaries with fibrin thrombi and fibromuscular hyperplasia of the lamina propria, as well as spindle cell proliferation).

Recognizing both types of lesion is important because of their specific management. PHG is reputed to respond to nonselective ß-blockers or transjugular intrahepatic portosystemic shunt (TIPS) if needed. Conversely, knowing it is a fixed lesion, GAVE do not respond to nonselective ß-blockers, but can be endoscopically eradicated by argon plasma coagulation, banding or radiofrequency ablation. Of course, mixed cases exist and need either haemodynamic and/or endoscopic therapies.[10]

In some cases, portal hypertension is associated with gastric polyps. Most of the time, these polyps are reddish with exudates on their top. Pathological analysis of these portal-hypertension-related gastric polyps reveals vascular dilations in the lamina propria with a small amount of lymphoplasmatocytic inflammation. These polyps can be associated with bleeding or anaemia, might also respond to treatment with nonselective ß-blockers, or can be removed if symptomatic.[11]

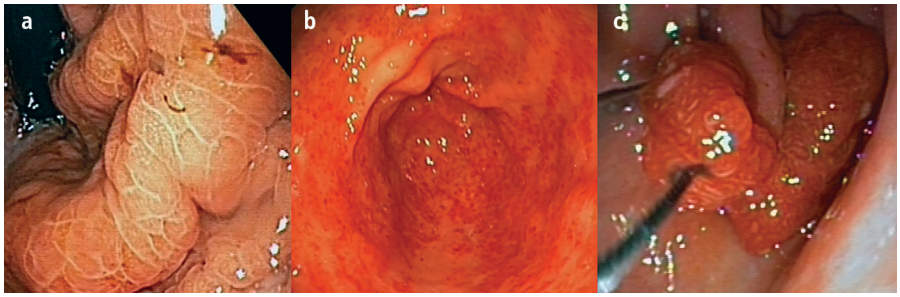

Figure 5 | Portal hypertensive gastric lesions. **a** | A subcardial vision of a patient with cirrhosis and portal hypertension showing the typical mosaic-like pattern of portal hypertensive gastropathy with red signs of diffuse tiny bleeding. **b** | Typical aspect of a GAVE in the antrum of a patient with cirrhosis with recurrent anaemia who was successfully treated by TIPS for refractory ascites and by argon plasma coagulation to eradicate GAVE lesions. **c** | A pyloric polyp with the typical appearance of a portal hypertensive gastric polyp (reddish with exudate) in a patient with cirrhosis. The polyp was resected for pathological analysis, showing dilated vessels. The patient had no recurrence of the gastric polyp at the follow-up examination.

Mistake 6 Choosing the wrong endoscopic treatment for early gastrointestinal neoplasia

Endoscopic diagnosis and management of early gastrointestinal neoplasia has dramatically progressed over the past 20 years. Nowadays, the optimal scenario for accurate diagnosis is good cleaning of the lumen of the GI tract segment, good characterization with an adequate endoscope with the addition of a virtual enhancement technique and/or chromoendoscopy (if needed), and, depending on the pit pattern and size, a choice of adequate resection techniques (figure 6).

In referral centres, we still take patients who were sent for surgery for benign or superficial neoplastic lesions that can be resected in a curative manner endoscopically, and we still see some patients with recurrence of partially resected lesions that were not treated adequately the first time. Moreover, many European endoscopists feel uncomfortable with pit pattern characterization.

European guidelines on endoscopic submucosal dissection (ESD) have now been published in *Endoscopy* and broad experience is also well described for endoscopic mucosal resection (EMR) of large colorectal lesions.[12,13] Briefly:

- For esophageal squamous cell carcinoma, EMR is reserved for lesions smaller than 10 mm if en-bloc resection can be foreseen. For larger lesions with a pit pattern and shape that favour a superficial lesion, en-bloc resection by ESD must be proposed.
- For the majority of visible lesions in Barrett oesophagus, EMR must be proposed as a staging, sampling resection method. ESD is reserved for lesions larger than 15mm, with poor lifting signs and lesions at risk of submucosal invasion.
- For gastric lesions, ESD is encouraged because of its better control of resection margins for lesions at low risk of lymph-node metastasis.
- For colorectal lesions, endoscopic resection by EMR is safe and most the time allows effective removal of the lesion. En-bloc resection by EMR or ESD (depending on the size) can be considered to remove lesions with high suspicion of limited submucosal invasion.

Mistake 7 Choosing an inadequate biliary stent when treating a benign biliary stricture or hilum tumours

Endoscopic retrograde cholangiopancreatography (ERCP) has evolved over the past 20 years towards being a purely therapeutic procedure. Stenting has also evolved with the successive availability of plastic stents, noncovered, partially covered and fully covered self-expandable metallic stents (SEMS). One major concern when performing stenting should be to not compromise any aspect of the patient's future outcome by implanting nonremovable stents. More and more pretherapeutic diagnostic tools, such as magnetic resonance cholangiopancreatography (MRCP) and endoscopic ultrasonography (EUS)/ fine-needle aspiration (FNA), are available to investigate the aetiology of a biliary stricture, for example. But, the definitive diagnosis still relies on pathology to demonstrate neoplasia. Even in case of diagnostic presumption, it must be kept in mind that placement of a noncovered SEMS in the biliary tree is a nonreversible treatment that has potential long-term complications in the case of incorrect diagnosis (figure 7).[14]

Along the same line, uncovered SEMS are the palliative treatment of choice for nonresectable hilum tumours, but they should never be implanted if nonresecability has not been confirmed. Indeed, multiple metallic stents at the hilum may render

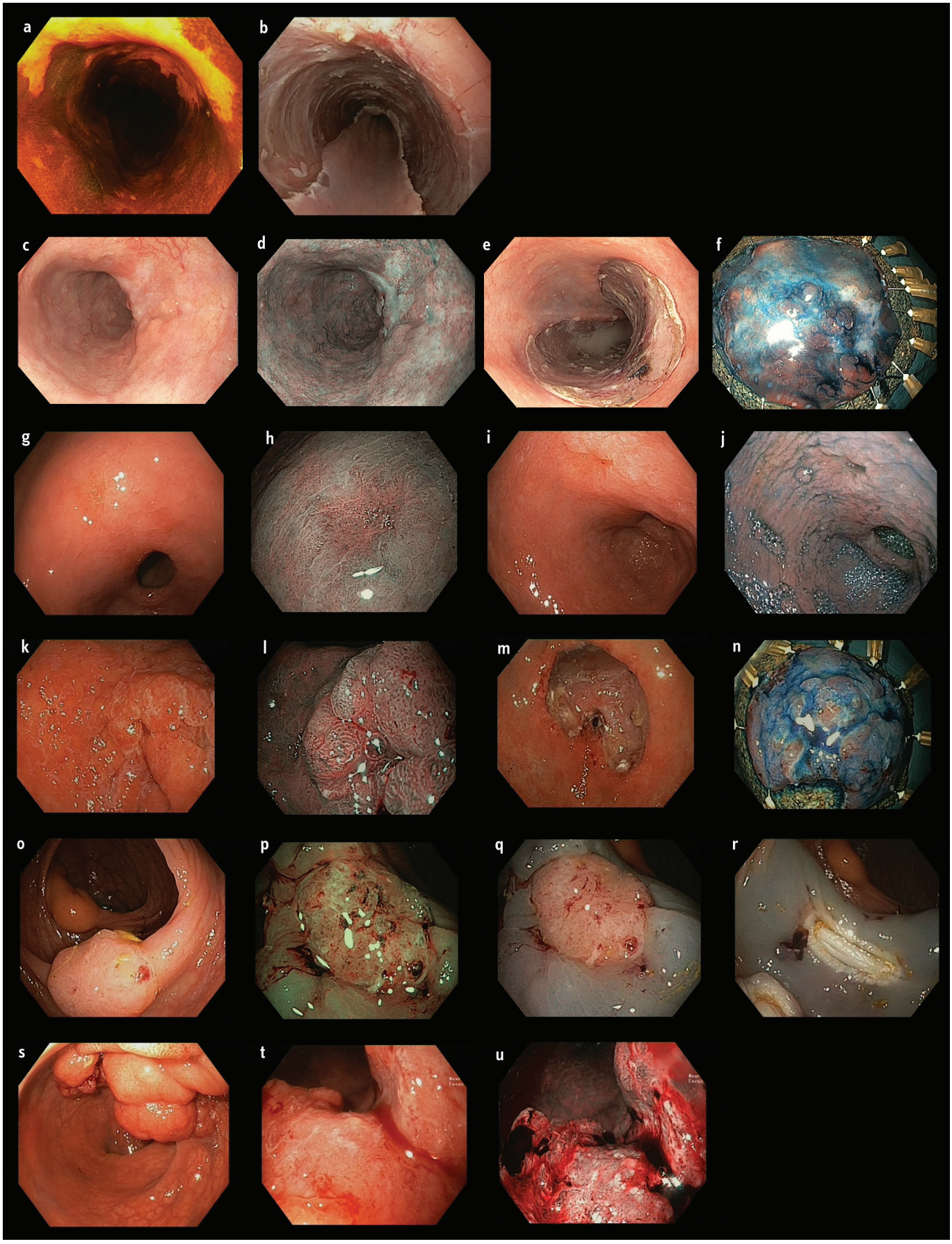
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Figure 6 | Examples of typical early neoplastic gastrointestinal lesions, their characterization and treatment modality. **a** and **b** | A 50-year-old male with an extensive flat Paris 0-IIb[16] squamous cell carcinoma was treated by ESD. Pathological analysis disclosed a pT1a well differentiated squamous cell carcinoma without any lymphovascular infiltration. Resection was R0 (clear vertical and horizontal margins). Endoscopic surveillance was proposed to the patient. **c–f** | A 73-year-old inoperable man with C12M17 Barrett oesophagus with a visible Paris 0-IIa malignant lesion of more than 3 cm was treated by ESD. The patient was asymptomatic after resection. Pathological analysis disclosed a well-differentiated adenocarcinoma infiltrating the submucosal layer (pT1b) with clear margins and no lymphovascular infiltration. In a patient not fit for surgery, this treatment benefitted his prognosis. **g–j** | Diagnosis of early gastric cancer is rare in Europe for many reasons, including low incidence and lack of awareness of how to recognize the lesions. The main issue is to clean the stomach to remove all the saliva, bubbles and a part of the mucus to be able to observe the mucosa carefully in patients at risk. Here is the case of a 73-year-old man known to have advanced OLGA stage IV metaplastic gastritis.[17] **g** | shows the aspect of the gastric mucosa after cleaning. A suspicious early lesion is seen as a little depressed reddish area in the angular incisure. **h** | Narrow-band imaging (NBI) illustrates the presence of a clear demarcation line with altered pit pattern in the middle part, surrounded by metaplastic tissue presenting the "light blue crest" sign.[18] **i** and **j** | Chromoendoscopy with acetic acid and indigo carmine increases the enhancement of the lesion to delineate it. A biopsy sample was taken, disclosing early gastric neoplasia with high-grade dysplasia, which is an indication for resection by ESD. **k–n** | A 83-year-old Portuguese patient presented with a severe diffuse metaplastic gastritis. A Paris 0-IIa prepyloric lesion was discovered on chromoendoscopy with acetic acid (**k**) and characterized by NBI (**l**), showing a clear demarcation line with altered pit pattern in the central part of the lesion, suggestive of early gastric cancer. A staging resection was performed by ESD (**m** and **n**). The pathological specimen disclosed a poorly differentiated adenocarcinoma invading the submucosa with lymphovascular infiltration (pT1b). Despite that the resection was R0, a complementary surgery was proposed to the patient, revealing 3 positive lymph nodes out of the 22 resected (pT1bN2). **o–r** | A 79-year-old woman was discovered to have a 15mm polypoid Paris 0-Is lesion in the right colon located on a fold (**o**). The lesion has a NICE III aspect on NBI suggestive of adenocarcinoma.[19] **p** | A good lifting was obtained with a 20% glycerol submucosal injection and en-bloc resection, which was mandatory in this case, was obtained by EMR. Pathological analysis disclosed a moderately differentiated adenocarcinoma infiltrating the submucosa and the superficial muscle layer (pT2Nx) with clear margins. A complementary surgery was discussed by multidisciplinary oncologic staff. **s–u** | A 56-year-old man was referred for rectal polyp resection. On EUS, the lesion was scored as uT1N0. On white light imaging, a Paris Is-IIa large adenoma infiltrating nearly half the circumference of the lower rectum and extending over 7cm was observed. With close visualization of the central area of the lesion, which was depressed compared with the rest of the polyp, using the near focus and NBI mode (11 b,c), it looked clear that the pit pattern was totally unstructured and staged as a Kudo VN pit pattern.[20] So, the patient was recused for ESD resection knowing the high suspicion for a deep submucosal infiltrating tumour. Thereafter, MRI disclosed a T3 lesion and the patient was referred for onco-surgical management.

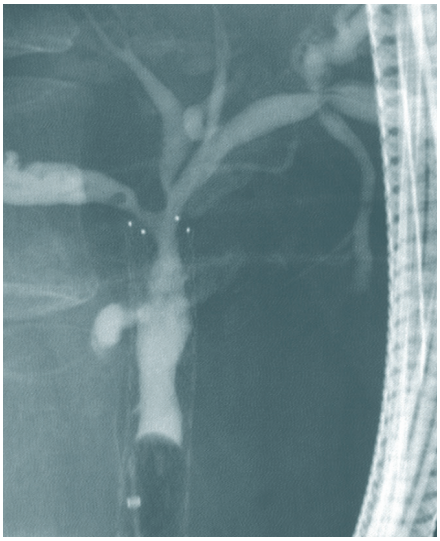

Figure 7 | The cholangiogram of a 74-year-old patient in whom a SEMS was placed for the treatment of a biliary stricture without an established malignant aetiology. At the patient's follow-up, a primary sclerosing cholangitis associated with quiescent inflammatory bowel disease (IBD) was diagnosed. The patient presented with recurrent suppurative cholangitis episodes due to obstruction at the hilum by hyperplasia and required more than 20 ERCP procedures in 5 years. In this case both the choice of stent (uncovered, which means nonremovable) and its length (extending to the hilum, thus compromising resectability in case of a tumour and even rendering a surgical anastomosis difficult) were inadequate.

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impossible extended right or left hepatectomy for curative resection of a hilar cholangiocarcinoma (figure 7).

Mistake 8 Missing a diagnosis of altered biliary anatomy during ERCP

Knowledge of anatomical variants of bile duct anatomy is essential for the practice of ERCP. The classical anatomy only represents 63% of the cases.[15] Most frequently, there is a bifurcation with the posterior segments implanted on the right hepatic duct. They are implanted at the hilum (trifurcation) in 10% of cases or on the left hepatic duct in 11% of the cases. In 4% of cases, the posterior segments are implanted lower on the common bile duct–below the hilum (2%) or directly on the cystic duct (2%). These situations are important to recognize in order to drain the liver adequately, to describe the anatomy for hepatobiliary surgery, and thus avoid potential complications (figure 8).[15]

Pre-therapeutic assessment of the biliary anatomy by MRCP helps when choosing the

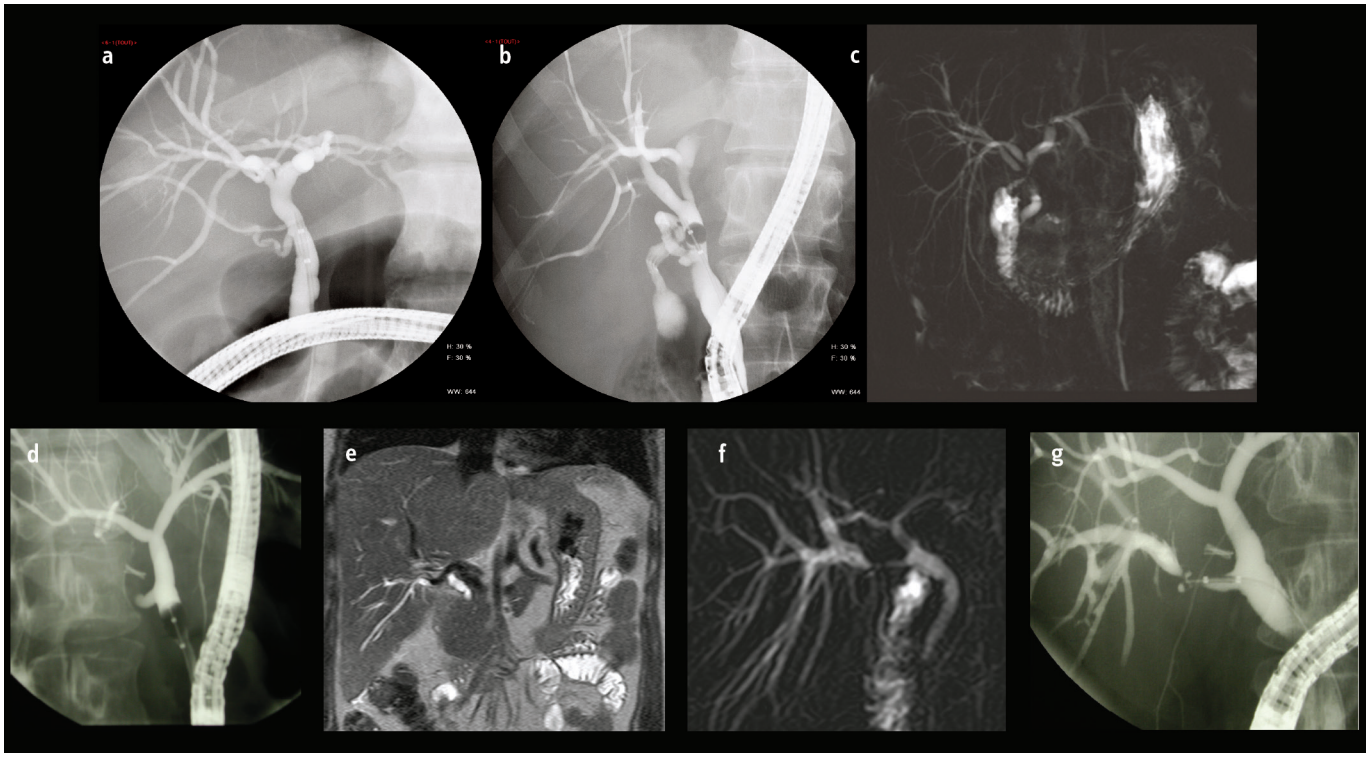

Figure 8 | Cholangiograms obtains by ERCP. **a** | Accessory duct for segment VII implanted on the common bile duct. **b** | Posterior segments implanted on the left hepatic duct. **c** | MRCP showing a hilar trifurcation (posterior segments implanted on the hilum). **d–g** | A 46-year-old patient developed right-upper quadrant pain and cholestasis after cholecystectomy. **d** | An initial ERCP showed an apparently normal hepatography, but, if you look carefully, posterolateral segments are missing on the cholangiogram. Either knowledge of the anatomy or MRCP (**e, f**) showed dilation of the posterolateral segments, implanted on the cystic duct. Then selective cystic duct cannulation and, by chance, endoscopic drainage are possible (**g**). A combined ERCP and percutaneous bile-duct access (using a TIPS set) to reconnect the excluded, dilated bile-duct segment to the common bile duct would have been an alternative treatment option.[21,22]

correct segment to catheterize and drain during the procedure.

Conflicts of interest: The authors declare there are no conflicts of interest.

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