# Endoscopic Resection of the Oesophagus and Oesophagogastric Junction

## Histopathology Reporting Guide

**Family/Last name**

**Given name(s)**

**Date of birth**

**Date of request**

**Accession/Laboratory number**

### CLINICAL INFORMATION

- Information not provided
- Relevant biopsy results, specify
- Previous history of cancers in the aerodigestive tract, specify
- Endoscopic location of the tumour, specify either measurement in mm from incisors or levels (upper/middle/lower)
- Clinical staging, specify level of involvement
- History of reflux and/or Barrett oesophagus
- Other, specify

### ENDOSCOPIC PROCEDURE

- Endoscopic mucosal resection (EMR)
- Endoscopic submucosal dissection (ESD)
- Other, specify

### SPECIMEN DIMENSIONS

(Record per specimen)

<table>
<thead>
<tr>
<th>mm</th>
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### MACROSCOPIC APPEARANCE

- No macroscopically detectable lesion
  - Polypoid
    - 0-Ip (protruded, pedunculated)
    - 0-Is (protruded, sessile; >2.5 mm above baseline)
  - Non-polypoid
    - 0-IIa (superficial, elevated; <2.5 mm above baseline)
    - 0-IIb (flat)
    - 0-IIc (superficial shallow, depressed)
    - 0-III (excavated/ulcerated)

### TUMOUR SITE

- Not specified
- Cervical (proximal) oesophagus
- Upper thoracic oesophagus
- Middle thoracic oesophagus
- Lower (distal) thoracic oesophagus
- Oesophagogastric junction (OGJ)
- Other, specify

Distance from epicentre/midpoint of tumour to OGJ

### TUMOUR DIMENSIONS

- Maximum tumour dimension
- Additional dimensions

### BARRETT MUCOSA

- Not identified
- Present

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**HISTOLOGICAL TUMOUR TYPE** (Note 8)  
(Value list from the World Health Organization Classification of Tumours of the Gastrointestinal Tract (2019))
- Squamous cell carcinoma
  - Conventional
  - Verrucous
  - Spindle cell carcinoma
  - Basaloid squamous cell carcinoma
- Adenocarcinoma
  - Tubular
  - Papillary
  - Mucinous
  - Poorly cohesive carcinoma
    - Signet ring
    - Non-signet ring
- Mucoepidermoid
- Adenosquamous carcinoma
- Adenoid cystic carcinoma
- Undifferentiated carcinoma
- Neuroendocrine neoplasms
- Neuroendocrine carcinoma
  - Small cell
  - Large cell
- Mixed neuroendocrine-non-neuroendocrine neoplasm (MiNEN)
- Other, specify

b  Neuroendocrine tumour is not covered in this dataset.

**DYSPLASIA** (select all that apply) (Note 9)
- Not applicable
- Type
  - Squamous
  - Columnar/Barrett
- Grade
  - Low grade
  - High grade
  - Cannot be assessed, specify

**HISTOLOGICAL TUMOUR GRADE** (Note 10)  
(Applicable to squamous cell carcinoma and adenocarcinoma)
- Not applicable
- GX: Cannot be assessed
- Grade 1 (G1): Well differentiated
- Grade 2 (G2): Moderately differentiated
- Grade 3 (G3): Poorly differentiated

**TISSUE LAYERS PRESENT** (select all that apply) (Note 11)
- Mucosa
- Glandular
- Squamous
- Mixed glandular and squamous
- Muscularis mucosae
- Deep muscularis mucosae
- Superficial muscularis mucosae
- Submucosa
- Muscularis propria

**EXTENT OF INVASION** (Note 12)
- Cannot be assessed
- No evidence of primary tumour (T0)
- Dysplasia with no invasion (Tis)
- Invasion into the lamina propria (T1a), specify depth of invasion
- Invasion into the muscularis mucosae (T1a)
- Invasion into the submucosa (T1b), specify depth of invasion
- Invasion into the muscularis propria (T2)

**LYMPHOVASCULAR INVASON** (Note 13)
- Not identified
- Present (select all that apply)
  - Small vessel (lymphatic, capillary or venular), specify lymphatic or vascular if possible
  - Large vessel (venous)

**PERINEURAL INVASON** (Note 14)
- Not identified
- Present

**MARGIN STATUS** (Note 15)
- Cannot be assessed, specify
- Invasive carcinoma
  - Not involved by invasive carcinoma
    - Distance of tumour from closest margin
    - Specify closest margin, if possible
  - Involved by invasive carcinoma (select all that apply)
    - Deep
    - Lateral
- Dysplasia
  - Not involved by dysplasia
    - Distance of dysplasia from closest margin
    - Specify closest margin, if possible
  - Involved by dysplasia
    - Squamous
      - Low grade
      - High grade
    - Columnar/Barrett
      - Low grade
      - High grade

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b  Measurement from the lamina propria of the epithelial cells.

c  Measurement from the lamina propria of the epithelial cells.

d  Measurement from lower border of muscularis mucosae.
COEXISTENT PATHOLOGY (Note 16)
- None identified
- Present, specify

ANCILLARY STUDIES (Note 17)
For neuroendocrine neoplasms only
- Not applicable
- Neuroendocrine markers (chromogranin A, synaptophysin, other), specify test(s) performed and result(s) if available

Ki-67 proliferation index: %
- Not performed
- Performed, specify

PATHOLOGICAL STAGING (UICC TNM 8th edition)* (Note 18)
(Applicable to specimens with sufficient tissue layers present)

TNM Descriptors (only if applicable)
- No adjuvant therapy
- y - post-therapy

Primary tumour (pT)
- TX Primary tumour cannot be assessed
- Tis Carcinoma in situ/high grade dysplasia
- T1 Tumour invades lamina propria, muscularis mucosae, or submucosae
  - T1a Tumour invades lamina propria or muscularis mucosae
  - T1b Tumour invades submucosa
- T2 Tumour invades muscularis propria

**Scope**

The dataset has been developed for the pathology reporting of endoscopic resection (ER) of premalignant and malignant lesions of the oesophagus and oesophagogastric junction (OGJ). Endoscopic biopsy specimens are also included. Surgically resected specimens are covered in a separate dataset.

Neuroendocrine carcinomas (NECs) and mixed neuroendocrine-non-neuroendocrine neoplasms (MiNENs) of the oesophagus are included.

Well differentiated neuroendocrine tumours (NETs), non-epithelial malignancies such as melanoma, and secondary tumours are excluded from this dataset.

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**Note 1 – Clinical information** *(Non-core)*

Clinical information can be provided by the clinician on the endoscopy report or the pathology request form. Pathologists may also search for additional information from previous pathology reports.

Relevant biopsy results include the presence of carcinoma, dysplasia, Barrett metaplasia, etc. Multiple tumours can occur in the oesophagus and especially in patients with a previous history of cancer e.g., carcinoma of hypopharynx.

Endoscopic location and information regarding the location of the tumour are an important guide. In addition, the depth of the invasion of early oesophageal cancer can be predicated by endoscopic appearance.¹

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**Note 2 – Endoscopic procedure** *(Core)*

Endoscopic resection (ER) is indicated in many early oesophageal cancers. Generally, ER for oesophageal cancer is limited to dysplasia and superficial mucosal cancers, whereas surgery is recommended for those with deep mucosal or submucosal invasion.

Endoscopic mucosal resection (EMR) is usually undertaken for mucosal lesions.² The complication rate for perforation for EMR is less than 2%.²

Endoscopic submucosal dissection (ESD) involves dissecting the submucosa to remove a larger oesophageal cancer and is technically more challenging. It allows for resection of lesions of much larger size but with higher complication rate.³,⁴

On pathological examination of a biopsy of early cancer, the presence of lymphovascular invasion, submucosal invasion, and poor tumour differentiation favour surgical treatment.⁵
Note 3 – Specimen dimensions (Core)

When the specimens are received piecemeal, they should be reconstructed for measurement purposes, if possible. The Oesophagus ER Dataset Authoring Committee recommended that the reporting of specimen dimensions should be a core element.

Note 4 – Macroscopic appearance (Non-core)

There is no evidence that macroscopic appearance has prognostic value in oesophageal cancer. However, the macroscopic appearance of the lesion, such as having an ulcerative appearance, could indicate the potential for a more advanced lesion.

The pathologist could also refer to the endoscopic appearance, if available, to compare the morphology (Figures 1 and 2).

An intramucosal cancer generally has a flat appearance (Paris classification 0-IIa, 0-IIb). By contrast, a submucosally invasive cancer often has an excavated (Paris classification 0-IIc, 0-III) and sometimes a polypoid morphology (Paris classification 0-I).\(^6\) In squamous cell carcinoma of the oesophagus, classification of surface vessels and intrapapillary capillary loops (IPCLs) also allows accurate assessment of invasion depth.\(^1\)

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**Figure 1: Neoplastic lesions with “superficial” morphology.** Reproduced with permission from Paris workshop participants (2003). The Paris endoscopic classification of superficial neoplastic lesions: esophagus, stomach, and colon: November 30 to December 1, 2002. *Gastrointest Endosc* 58(6 Suppl):S3-43.\(^6\)
Figure 2: Schematic representation of the major variants of type 0 neoplastic lesions of the digestive tract: polypoid (Ip and Is), non-polypoid (Iia, Iib, and Iic), non-polypoid and excavated (III). Terminology as proposed in a consensus macroscopic description of superficial neoplastic lesions. Reproduced with permission from Paris workshop participants (2003). The Paris endoscopic classification of superficial neoplastic lesions: esophagus, stomach, and colon: November 30 to December 1, 2002. Gastrointest Endosc 58(6 Suppl):S3-43.

Note 5 – Tumour site (Core & Non-core)

The location of the tumour is important for staging of oesophageal cancer.

The location of a cancer is based on endoscopic examination and landmarks, therefore, clinical information provided by surgeon or endoscopist is critical.

The anatomical subdivisions of the oesophagus are outlined below (Figure 3):

- The cervical (proximal) oesophagus begins at the lower end of the pharynx (at the level of the 6th vertebra or lower border of cricoid cartilage) and extends to the thoracic inlet (suprasternal notch); 18 centimetres (cm) from the incisors.
- Upper thoracic extends from the thoracic inlet to the level of tracheal bifurcation; 18-23 cm from the incisors.
- Middle thoracic extends from the tracheal bifurcation midway to the OGJ; 24-32 cm from the incisors.
- Lower (distal) thoracic extends from midway between the tracheal bifurcation and gastroesophageal junction to the OGJ, including the abdominal oesophagus; 32-40 cm from the incisors.
Figure 3: Anatomic subdivisions of the oesophagus. Used with permission of the American College of Surgeons, Chicago, Illinois. The original source for this information is the AJCC Cancer Staging Manual, Eighth Edition (2016) published by Springer Science+Business Media.7

A description of the tumour site is ideally provided by the surgeon and should be documented by the pathologist. In addition, specific observations should be recorded by the pathologist which may help establish the exact site of origin of the tumour. Where possible, the pathologist should record the maximum longitudinal dimension of the tumour mass, the distance of the tumour midpoint from the OGJ, and the relative proportions of the tumour mass located in the oesophagus and in the stomach.

Pure anatomical classification of the tumour site of origin can be defined in a number of different systems.

Siewert and colleagues define adenocarcinomas involving the OGJ based upon location of the centre of the tumour into 3 categories as follows:8

Type I: Carcinoma of the distal oesophagus, with or without infiltration of the OGJ from above
Type II: True carcinoma of the gastric cardia (proximal stomach), arising from the cardiac epithelium or short segments with intestinal metaplasia at the OGJ
Type III: Subcardial gastric carcinoma, which infiltrates the OGJ and distal oesophagus from below.

The American Joint Committee on Cancer (AJCC) and College of American Pathologists (CAP) define the OGJ as the junction of the tubular oesophagus and the stomach, irrespective of the type of epithelial lining of the oesophagus.7,9

The AJCC 8th edition Staging Manual7 also defines tumours involving the OGJ as those with a midpoint within the proximal 20 millimetres (mm) of the cardia/proximal stomach and are staged as oesophageal cancers. Cancers are staged as stomach cancer when the epicentre is more than 20 mm distal from the OGJ, even if the OGJ is involved.7
Some proximal stomach tumours which appear to be of gastric origin, under the AJCC 8th edition classification, may be classified as tumours of the oesophagus and OGJ somewhat artificially and thus reported using the oesophageal dataset. When reporting such tumours, it should be noted that the tumour may have arisen within the stomach.

If there are synchronous primary lesions (i.e., two or more individual tumours), separate datasets should be used to record the tumour site and all following elements for each primary tumour.

**Note 6 – Tumour dimensions (Core & Non-core)**

If no tumour is macroscopically visible, or for small tumours where the macroscopic dimensions may not be accurate then the microscopic dimensions should be documented.

If the specimen is fragmented, measurements of the reconstructed tumour should be estimated, where possible. Otherwise, the clinical and/or radiological measurements should be used.

**Note 7 – Barrett mucosa (Core)**

The presence of intestinal and foveolar dysplasia points to the aetiology of the oesophageal adenocarcinoma.

**Note 8 – Histological tumour type (Core)**

Pathological staging is different for the two major groups of oesophageal carcinomas, adenocarcinoma and squamous cell carcinoma. It is important to refer to the current World Health Organization (WHO) classification of the different oesophageal malignant neoplasms.

Adenoid cystic carcinoma, undifferentiated carcinoma or NEC with an adenocarcinoma component, follow the adenocarcinoma stage grouping. There is no definite evidence for whether the staging of adenosquamous carcinoma or mucoepidermoid carcinoma should follow that of squamous cell carcinoma or adenocarcinoma staging groups.

For adenocarcinoma, there are different histological patterns. In most instances, they could be grouped either into tubular, papillary and mucinous patterns. In rare circumstances, the tumour could be poorly cohesive and have either signet ring or non-signet ring pattern.

In MiNENs of the oesophagus, the neuroendocrine component is nearly always NEC.
Note 9 – Dysplasia (Core)

There are two types of dysplasia, squamous dysplasia and columnar (either Barrett or non-Barrett) dysplasia.

In the current WHO classification, both squamous and Barrett dysplasia are classified using a two-tiered system, high and low grade.\(^\text{11}\)

Columnar dysplasia is mostly Barrett dysplasia. The presence of Barrett dysplasia supports oesophageal origin of an adenocarcinoma.

The term Barrett dysplasia in the WHO classification is adopted because of the aetiological link with Barrett oesophagus. However, it is noted that rare cases of oesophageal adenocarcinoma may not arise from Barrett dysplasia. For instance, some rare adenocarcinoma of the mid oesophagus have no relationship with Barrett dysplasia.\(^\text{11}\)

Oesophageal columnar neoplasia is broadly divided into gastric, intestinal and mixed (hybrid) types, based on morphological and immunohistochemical features. The clinical significance of this division is yet to be determined and is not needed for routine clinical care.

Over the past 10 years or more, there has been an important shift from surgery towards ablation for the treatment of Barrett oesophagus in patients with high grade dysplasia.\(^\text{11}\) Whether confirmed low grade dysplasia justifies invasive management, is currently a controversial issue.\(^\text{11}\)

↑ Back

Note 10 – Histological tumour grade (Core)

Grade (differentiation) of the tumour contributes to pathological staging.\(^\text{7}\)

The 5\(^{\text{th}}\) edition of WHO classification has defined the morphological criteria for grading of adenocarcinoma and squamous cell carcinoma.\(^\text{11}\)

In adenocarcinoma, grade 1 is defined as adenocarcinoma with >95% of the carcinoma with well-formed glands; grade 2 with 50% to 95% with well-formed glands; grade 3 is <50% with glandular formation.\(^\text{12}\)

In squamous cell carcinoma, grade 1 to grade 3 depends on the amount of keratin pearls, cytological atypia, mitotic activity and proportion of basaloid cells.\(^\text{10}\)

↑ Back
Note 11 – Tissue layers present (Core)

Reporting of the tissue layers present in the specimen is important as it provides context for the assessment of extent of invasion. For example, it is not possible to assess submucosal invasion if an ER specimen consists only of the mucosa.

It is worth noting that muscularis mucosae often duplicates, and this should be considered on assessment of the tissue present and the level of invasion.

In Barrett oesophagus, in addition to the original muscularis mucosae, a second (‘neo’) muscularis mucosae is often formed. The original muscularis mucosae is defined as the deep muscularis mucosae, and the newly derived muscularis mucosae is defined the superficial muscularis mucosae.

Note 12 – Extent of invasion (Core)

The Union for International Cancer Control (UICC\(^{13}/\text{AJCC}\)\(^{7}\) 8\(^{th}\) edition Staging Manuals divide T stage into T1a and T1b. T1a refers to invasion into lamina propria or muscularis mucosae whereas T1b involves the submucosa.\(^7\) Thus, the depth of invasion which is the T staging criteria, must be recorded accurately.

It is also useful to measure the depth of invasion from the basement membrane of the epithelial layer and invasion to the submucosa (in mm).

In addition, the extent of invasion has been associated with lymphovascular invasion and recurrence. For both glandular and squamous malignancies, there are efforts to further subdivide the level of invasion. However, there is lack of multicentred studies to confirm the need of these subdivisions and to evaluate the best system to use.

The following are the systems commonly employed for reference for optional use:

For adenocarcinoma and high grade Barrett dysplasia
In these malignancies, the Barrett muscularis propria is duplicated (Figures 4 and 5, Table 1).\(^7,14-16\) There is a proposal to subdivide the involvement of muscularis mucosae into 3 classes (M2 to M4) as follows:

- Cannot be assessed
- High grade dysplasia (M1) Tis
- Invasion into lamina propria (M2, T1a)
- Invasion into muscularis mucosae (Inner duplicated layer) (M3, T1a)
- Invasion into muscularis mucosae (Outer duplicated layer) (M4, T1a)
- Invasion into submucosa (T1b)
- Invasion into muscularis propria (T2)
Figure 4: Histo-anatomical layers in oesophageal squamous (M1-M3) and Barrett mucosa (M1-M4). Permission courtesy of Cord Langer.

Table 1: Intramucosal carcinoma (T1a) subclassification schemes.\textsuperscript{15-17}

<table>
<thead>
<tr>
<th>Depth of invasion</th>
<th>Vieth et al 2005\textsuperscript{14}</th>
<th>Westerterp et al 2005\textsuperscript{16}</th>
<th>Kaneshiro et al 2011\textsuperscript{15}</th>
<th>AJCC 2017\textsuperscript{18}</th>
</tr>
</thead>
<tbody>
<tr>
<td>None - Tis, high grade dysplasia (HGD)</td>
<td>HGD</td>
<td>m1</td>
<td>HGD</td>
<td>Tis</td>
</tr>
<tr>
<td>Tumour cells invade into lamina propria (LP) beyond the basement membrane</td>
<td>m1</td>
<td>m2</td>
<td>LP</td>
<td>T1a</td>
</tr>
<tr>
<td>Tumour cells invade inner duplicated muscularis mucosae (IMM)</td>
<td>m2</td>
<td>m2</td>
<td>IMM</td>
<td>T1a</td>
</tr>
<tr>
<td>Tumour cells in the space between the duplicated muscularis mucosae and original muscularis mucosae, i.e., between muscularis mucosae (BMM)</td>
<td>m3</td>
<td>m2</td>
<td>BMM</td>
<td>T1a</td>
</tr>
<tr>
<td>Tumour cells into outer original muscularis mucosae (OMM)</td>
<td>m4</td>
<td>m3</td>
<td>OMM</td>
<td>T1a</td>
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For squamous cell carcinoma and high grade squamous dysplasia

For these malignancies, Japanese pathologists have proposed a different sub-division of levels of invasion as follows:

- T1a-EP
- T1a-LPM
- T1a-MM
- T1b-SM1
- T1b-SM2
- T1b-SM3

pT1 of intramucosal cancer is assessed in the three stages, including pT1-EP (epithelium), pT1a-LPM (lichen planus mucosae) and pT1a-MM (muscularis mucosae) (Figures 6 and 7).

When cancer remains in the mucosal layer, the depth of invasion is subclassified into three levels, pT1a-EP (cancer cells remain in the columnar epithelial layer or the superficial muscularis mucosae), pT1a-LPM (cancer cells exceed the superficial muscularis mucosae but do not reach the deep muscularis mucosae) and pT1a-MM (cancer cells invade the deep muscularis mucosae).

For cancer that invades the submucosa, the submucosa is divided into three equal parts to express the depth of invasion under microscopic observation - the top layer, middle layer, and bottom layer are pSM1, pSM2, and pSM3, respectively.

In a cancer that invades beyond the muscularis mucosae of an endoscopic resection case, the entire submucosal layer cannot be observed. Therefore, the depth of invasion from the lower end of the muscularis mucosae should be described using measured values. The subclassification of pT1b is pT1b-SM1 for cancer cell invasion up to 200 micrometres (μm) and pT1b-SM2 for cancer cell invasion exceeding 200 μm.
One of the rationales for this subdivision is that the risk of lymph node metastasis is shown to be related to the invasive depth for endoscopic resection cases.\textsuperscript{19,20}

\textbf{Figure 6:} pT1 of intramucosal squamous cancer is assessed in the three stages: pT1-EP (epithelium), pT1a-LPM (lamina propria mucosae) and pT1a-MM (muscularis mucosae). The subclassification of pT1b is: pT1b-SM (submucosa) 1 for cancer cell invasion up to 200 \( \mu \)m and pT1b-SM2 for cancer cell invasion exceeding 200 \( \mu \)m; MP (muscularis propria). Modified with permission from Japan Esophageal Society (2017). Japanese Classification of Esophageal Cancer, 11\textsuperscript{th} Edition: Part I. Esophagus 14:1–36.\textsuperscript{21} Additional permission courtesy of Satoshi Fujii. Copyright © The Author(s) 2016. Open Access - This content is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/)
Figure 7: The system of subdivision applied to both squamous and glandular malignancies. EP: epithelium; LPM: lamina propria; MM: muscularis mucosae; MP: muscularis propria; SM: submucosa; SMM: superficial muscularis mucosae; DMM: deep muscularis mucosae. Modified with permission from Japan Esophageal Society (2017). Japanese Classification of Esophageal Cancer, 11th Edition: Part I. Esophagus 14:1–36. Additional permission courtesy of Satoshi Fujii. Copyright © The Author(s) 2016. Open Access - This content is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/)

Note 13 – Lymphovascular invasion (Core)

Lymphovascular invasion is a known poor prognostic factor in oesophageal carcinomas and is designated a core element.

The value of distinguishing lymphatic from venous invasion has not been investigated. Venous invasion is important for extramural veins.

It is recommended that if possible, they should be reported separately, to provide data for further analysis. If this is not possible, report it as lymphovascular invasion.
Note 14 – Perineural invasion (Non-core)

Perineural invasion is an uncommon finding in endoscopic resection specimens and more studies are needed to validate its impact, therefore it is designated as a non-core parameter.

Note 15 – Margin status (Core)

Where there are multiple tumours, none of which involve a margin, the distance from the lesion nearest to the lateral/radial resection margin should be measured.

If the specimen is received piecemeal, the status of the margins may not be assessable.

For multifocal tumours, the presence of positive margin in any tumours should be indicated as “positive”, and the closest margin can be measured from any tumours in the specimen.

Note 16 – Coexistent pathology (Non-core)

Common coexisting pathology other than Barrett oesophagus may include scar tissue, leiomyoma, squamous papilloma, etc.

Note 17 – Ancillary studies (Core & Non-core)

For mixed neuroendocrine-non-neuroendocrine carcinomas (MiNECs) and NECs, “Ancillary studies” is a core element to report neuroendocrine marker expression, whereas this element is non-core for other types of oesophageal cancer. Neuroendocrine neoplasms are classified into NETs, NECs and MiNECs. NETs are graded 1-3 using the mitotic count and Ki-67 proliferation index but pure NETs are not considered within the scope of this dataset. Most NECs show marked cytological atypia, brisk mitotic activity, and are subclassified into small cell and large cell subtypes. NECs are considered high-grade by definition. MiNECs are usually composed of a poorly differentiated NEC component and an adenocarcinoma component. If a pure or mixed NEC is suspected on morphology, immunohistochemistry is required to confirm neuroendocrine differentiation, usually applying synaptophysin and chromogranin A as a minimum. p53 may be used to assess the presence of Barrett dysplasia in selected cases, though it is more useful in the endoscopic biopsy setting rather than for endoscopic resection.
Pathological staging is the most important factor to predict the survival of patients with oesophageal carcinomas.

In the 8th edition UICC Staging Manual, there is only one staging protocol for both squamous cell carcinoma and adenocarcinoma. The staging grouping is different from that without therapy. The grade of carcinoma is not a criterion for the stage grouping.

For endoscopic resection only T1 and T2 are used because of the absence of muscularis propria and adventitia.

References


