

PROPHYLACTIC OESOPHAGECTOMY: WHO, WHEN, HOW MUCH AND WHAT LONG- TERM OUTCOME

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INTRODUCTION: CARCINOMA OESOPHAGUS

- Two types of Ca Oesophagus
- Squamous Cell Carcinoma
 - disease of the poor
 - many risks factors but smoking, drinking and micronutrient deficiency important
 - Only genetic predisposition in rare palmar-plantar hyperkeratosis (Tylosis)
- Adenocarcinoma
 - Disease of life style of the wealthy
 - Major risk factor is GORD and Barrett's Oesophagus

BARRETT'S OESOPHAGUS

- Columnar metaplasia of oesophagus
- Associated with GORD
- Columnar metaplasia→dysplasia
- Intestinal type metaplasia more prone to dysplasia
- High Grade dysplasia is premalignant

Adenocarcinoma Risk in Barrett's Oesophagus

- Life time risk of ADCO 10-46%
- 50-86% ADCO originate from Barrett's
- 50-66% High Grade Barrett's show early invasive malignancy

Screening in Barrett's Oesophagus

- Systematic screening for oesophageal columnar metaplasia
- Target affluent males (especially White)
- Screening oesophagoscopy is cost –effective (cf breast cancer screening)

Management of Barrett's Oesophagus (I)

- AIM
 - Reverse Barrett's Oesophagus
 - Prevent progression of metaplasia to dysplasia
 - Arrest progression of dysplasia to ADCO
- Although intestinal metaplasia carries highest risks, all columnar metaplasia can be dysplastic

MANAGEMENT OF BARRETT'S OESOPHAGUS (II)

- Medical Prophylactic Treatment
 - Life-long acid suppression treatment with PPI or H₂R antagonist have not been shown to prevent ADCO
 - Selective COX-2 inhibitors no better than placebo
- Nonsurgical Endoscopic Prophylaxis
- Surgical Prophylaxis

Non-Surgical Endoscopic Treatment of Barrett's Oesophagus

- Nd-YAG laser photocoagulation
- Argon plasma coagulation
- Multipolar electrocautery
- Photodynamic therapy
- Cryotherapy ablation
- Radio frequency ablation

Main drawback is that there is **no tissue to examine for malignant change**

Surgical Treatment of Barrett's Oesophagus

- Anti-reflux surgical procedures e.g. Nissen Fundoplication reduce progression of BO to dysplasia
- Many anti-reflux procedures fail to prevent reflux in the long run
- Finnish study showed that anti-reflux procedure did not prevent development of ADCO

TREATMENT OF BARRETT'S DYSPLASIA

- Barrett's high grade dysplasia is precancerous (or Tis)
- Treatment aims to obliterate dysplastic mucosa
- Problem is difficulty in determining which dysplastic focus is already invasive
- Any ablative therapy **must** therefore **avail material for histology**
- Ablation of low grade dysplasia is controversial – most would observe

Classification of Early Cancer of Oesophagus

- Tis or Carcinoma –in-situ (high grade dysplasia)
- T1 carcinoma limited to submucosa
- T2 carcinoma onto muscularis propria
- T3 is through adventitia
- T4 is onto adjacent organs e.g. peri cardium, trachea
- Tis and T1 further classified thus:
 - m1 =epithelial (intra-epithelial) cancer
 - m2 = mucosal cancer invading into lamina propria
 - m3= mucosal cancer invading muscularis mucosa
 - sm = submucosal cancer

Once **basement membrane has been breached lymphatic spread** may be found

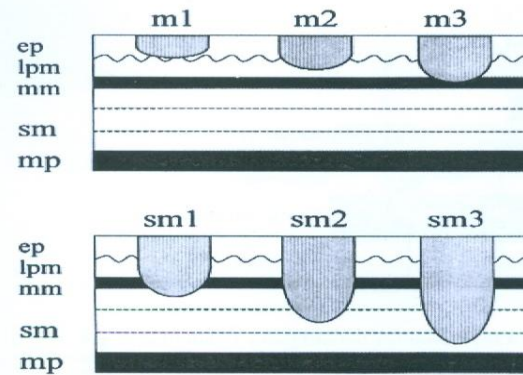


Fig 1. Superficial esophageal carcinoma was subclassified as follows: (m1) intraepithelial carcinoma or cancer that barely breaks the basement membrane; (m3) carcinoma that is extremely close to or infiltrates the lamina muscularis mucosae; (m2) lesions between m1 and m3; (sm1) carcinoma limited to the upper one-third of the submucosal layer; (sm2) carcinoma limited to the middle one-third of the submucosal layer; (sm3) carcinoma limited to the lower one-third of the submucosal layer.

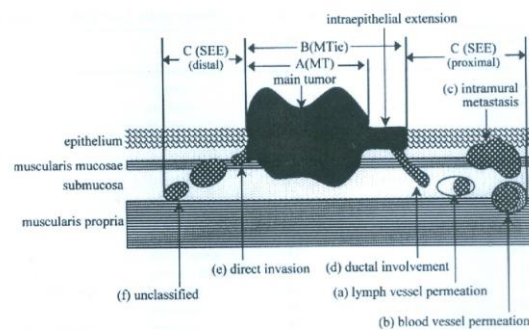
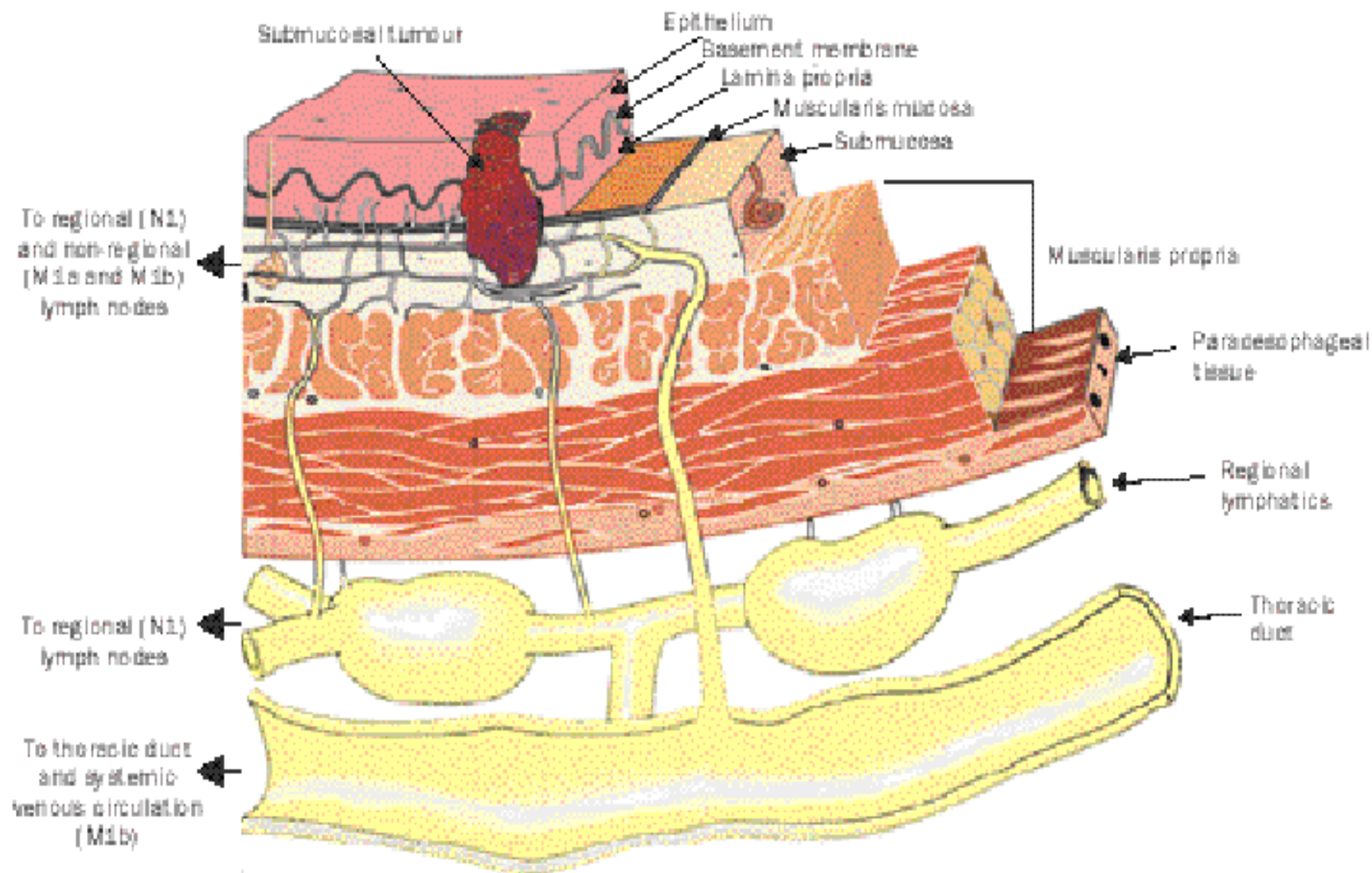


Fig 2. Schema of subepithelial extension (SEE) and measurement of tumor diameter and SEE. All of the cancer nests existing under the normal esophageal or gastric epithelium are defined as SEE. Elements of SEE are classified as (a) lymph vessel permeation, (b) blood vessel permeation, (c) intramural metastasis, (d) ductal involvement, (e) direct invasion, and (f) cancer nest which cannot judge what element to be. The diameter of only (A), the main tumor (MT), (B), the tumor diameter with intraepithelial extension (MTie), and (C), the length of SEE were measured.

Lymphatic Spread of Early Oesophageal Cancer

- Once the basement membrane is breached lymphatic invasion is possible
- Oesophageal cancer spreads early into lymph nodes
 - Local regional
 - “distant” LN either cephalad or caudal or both
- Early lymph node metastases difficult to detect
- Endoluminal ultrasound best imaging modality but not infallible
- Once invasive cancer has breached basement membrane lymph node dissection is mandatory



Lymphatic anatomy of the oesophagus

There are three routes of lymphatic flow and potential lymphatic and systemic metastases.

Options for extirpation of Barrett's Oesophagus

- Nonsurgical BO ablation do not avail tissue for histology therefore NOT an option
- Endoscopic Mucosal Resection
 - drawback is no lymph node assessment possible
 - patients with cancer beyond basement membrane must be subjected to formal surgery
- Formal Oesophagectomy
 - many surgical options
 - Traditional transthoracic and newer transhiatal are equivalent
 - New minimally invasive techniques via thoracoscopic/laparoscopic approaches need further evaluation
 - Limited segmental oesophagectomy should be reserved for otherwise surgically unfit patients

Long Term Survival of Early ADCO after Oesophagectomy

- 5yr Survival in Early ADCO
 - surveillance group=86%
 - non-surveillance =56%

Conclusion

- Prophylactic prevention of invasive ADCO require extirpation of high grade dysplastic oesophageal mucosa
- Histological examination of the dysplastic mucosa mandatory to detect early invasive ADCO
- Endoscopic mucosectomy suitable in expert hands
- Invasive carcinoma needs formal oesophagectomy.