Eosinophilic esophagitis

Bible class 21.04.2021

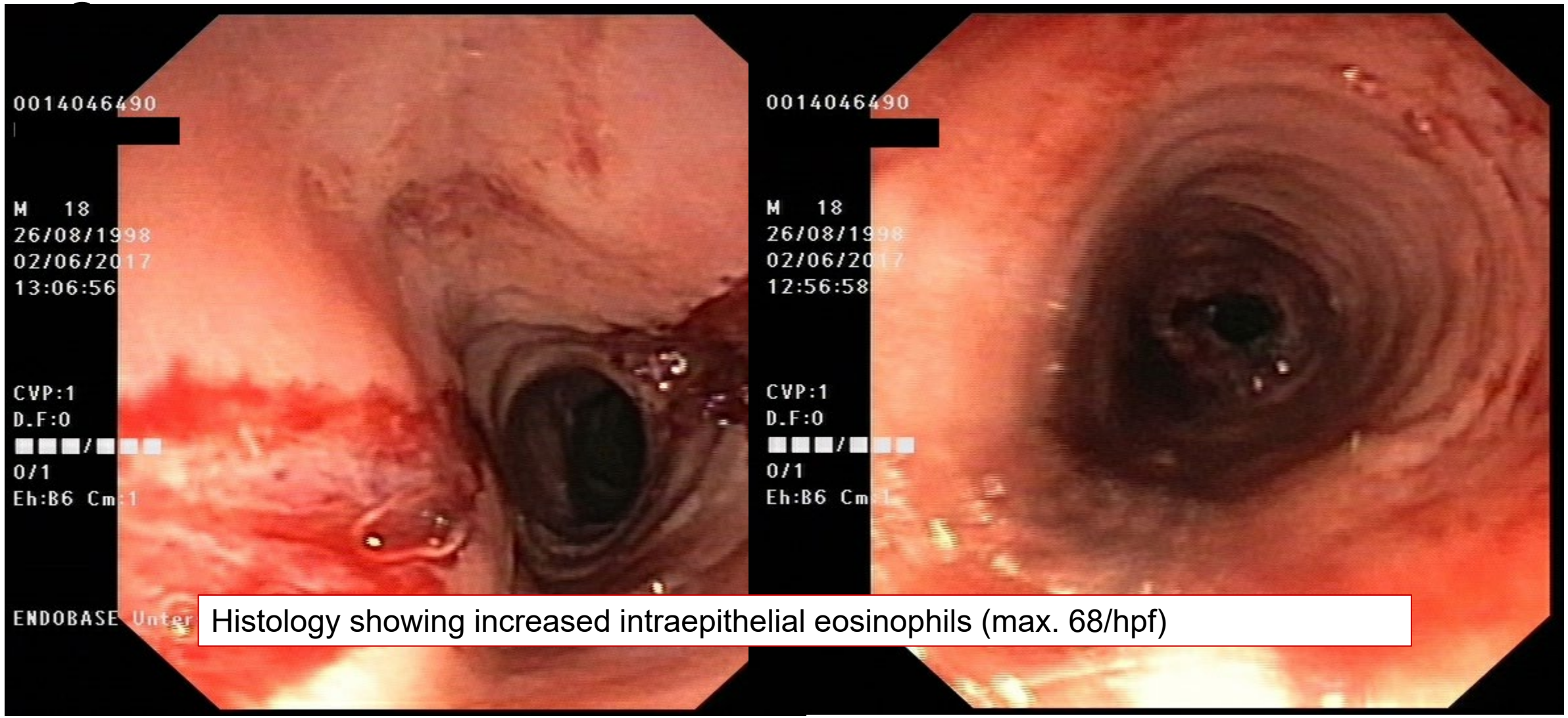
Dr. med. Matthias Knecht

Case

- Mr. B. J., 26.08.1998

- Presenting to the endoscopy unit with recurring food impaction
- Since childhood slow eater and often retrosternal pressure whilst eating

- Further diagnoses
  - Asthma bronchiale
  - Allergy to soy and eggs



Histology showing increased intraepithelial eosinophils (max. 68/hpf)

Case

- Esophageal manometry
  - Hypertensive LES, ineffective motility, high grade bolus transport disorder (liquid and viscous)

- 24h-impedance-pH-metry
  - Very little acid exposure, normal amount of reflux episodes. SAI negative for dysphagia

- Barium swallow
  - Normal passage, no strictures

Case

- Diagnosis of eosinophilic esophagitis
- Treatment with high dose PPI

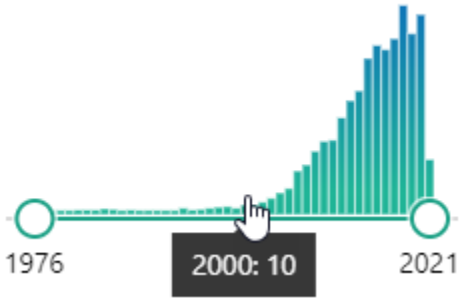
Definition of EoE

- Chronic, local immune-mediated esophageal disease
- Symptoms related to esophageal dysfunction
- Histologically eosinophil-predominant inflammation
- Exclusion of other causes of eosinophilia (local and/or systemic)

History of eosinophilic esophagitis (EoE)

- First description of a case in 1970
- Recognition as a disease in the early 90ies
  - Alex Straumann et al, Switzerland (Spital Olten!). Published 1994 in swiss me
  - Stephen Attwood et al, USA. Published 1993 in Digestive Disease Science

- Increase in studies after 2000



- First guidelines in 2007

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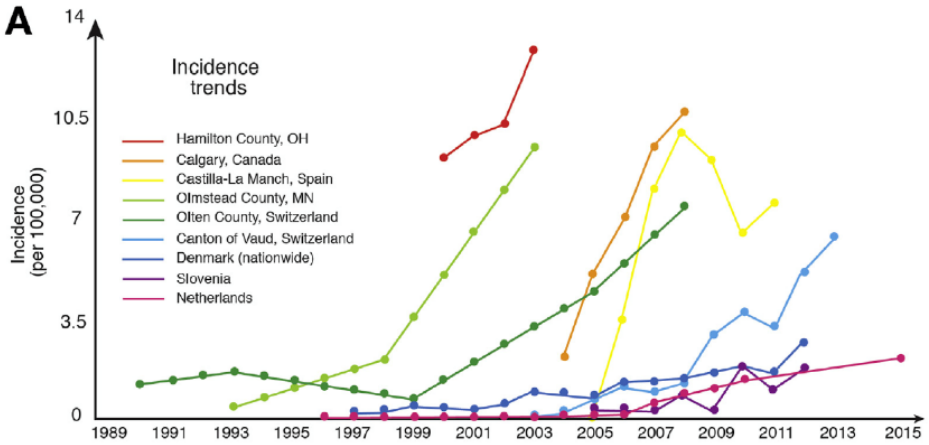
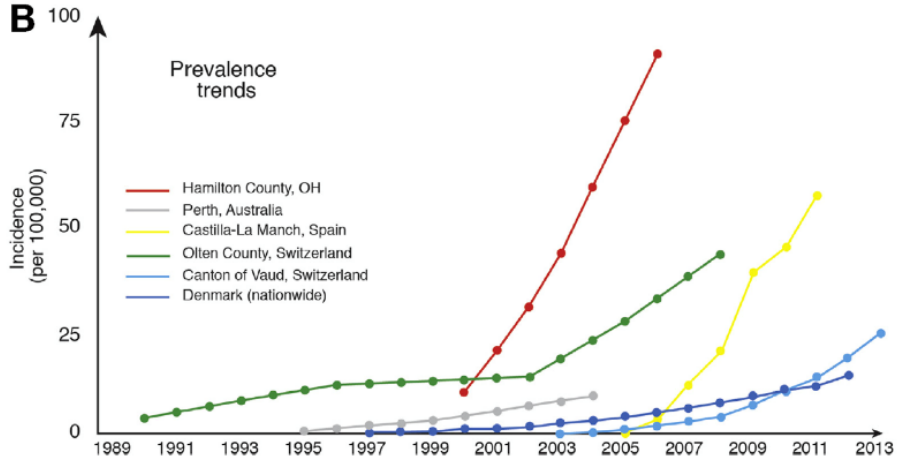
Epidemiology

- Prevalence 10 - 81 cases per 100’000 persons

- Increasing incidence
  - 2007 3 cases per 100’000 persons[1]
  - 2016 13.7 cases per 100’000 persons[1]
- Switzerland?
  - Prevalence 24.1-42.8 cases per 100’000 [2, 3]
  - Incidence increasing from 0.16 to 6.3 cases per 100’000 (Factor 40 from 1993 to 2013) [2]



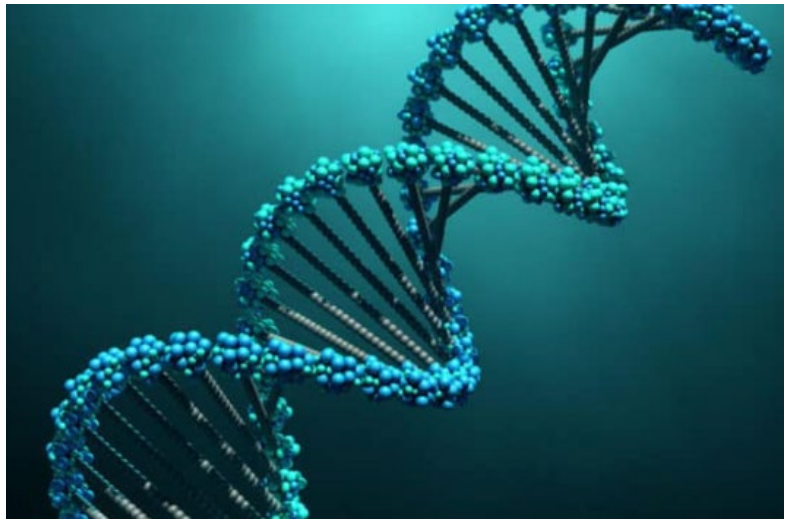


[1] Molina-Infante J et al. Rising incidence and prevalence of adult eosinophilic esophagitis in midwestern Spain (2007–2016). United European Gastroenterol J. 2018 Feb; 6(1): 29–37.
[2] Schoepfer A. Escalating Incidence of Eosinophilic Esophagitis in Canton of Vaud, Switzerland, 1993 to 2013: a population-based study
[3] Giriens B et al. Escalating incidence of eosinophilic esophagitis: a 20-year prospective, population-based study in Olten County, Switzerland. J Allergy Clin Immunol 2011; 128:1349.

Classical patient?

- Predominantely men (ratio 3:1)
- Median age around 30y, rarely old age
- Caucasian
- Urban-rural, north-south/west-east gradient
- History of atopy

Risk factors



- **Genetics and familial clustering**
  - Thymic stromal lymphopoetin (TSLP) -> Th2 differentiation
  - CCL26 -> Production of eotaxin-3
  - CAPN14 -> IL-13 induced proteolytice enzyme, esophagus specific

Often in atopics

EoE-specific

- High risk of genetic inheritance
  - Relative risk up to 64

Collins MH. Gastroenterol Clin North Am. 2014 Jun

Table 1. EoE Genetic Risk Loci (Statistically Significant and Replicated)

Genetic risk loci	Genes encoded	Odds ratio for most associated SNP at each locus	Genetic mechanism	Pathogenic mechanism
2p23	*CAPN14*	1.98	Promoter variant leads to genotype-dependent expression of CAPN14, likely involving epigenetic mechanism	CAPN14 is a proteolytic enzyme specific to the esophagus that is induced by IL-13 and involved in epithelial homeostasis and repair
5q22	*TSLP* *WDR36*	0.74	Multiple risk alleles associated with genotype-dependent expression of TSLP	TSLP induces Th2 cell development and activates eosinophils and basophils
11q13	*LRRC32* *EMSY*	2.49	Not yet described	LRRC32 is a TGF-beta binding protein EMSY is involved in transcriptional regulation
12q13	*STAT6*	1.5	Not yet described	STAT6 is a downstream signaling mediator of IL-4Rα and important for Th2 development
19q13	*ANKRD27* *PDCD5* *RGS9BP*	1.6	Not yet described	ANKRD27 inhibits the SNARE complex PDCD5 is involved in apoptotic pathways RGS9BP is not expressed in the esophagus or by immune cells

NOTE. Risk shown is positive and hence adjusted for being a common or rare allele.
EoE, eosinophilic esophagitis; SNP, single-nucleotide polymorphism; TSLP, thymic stromal lymphopoietin.

Table 3. Mendelian Diseases Associated With EoE

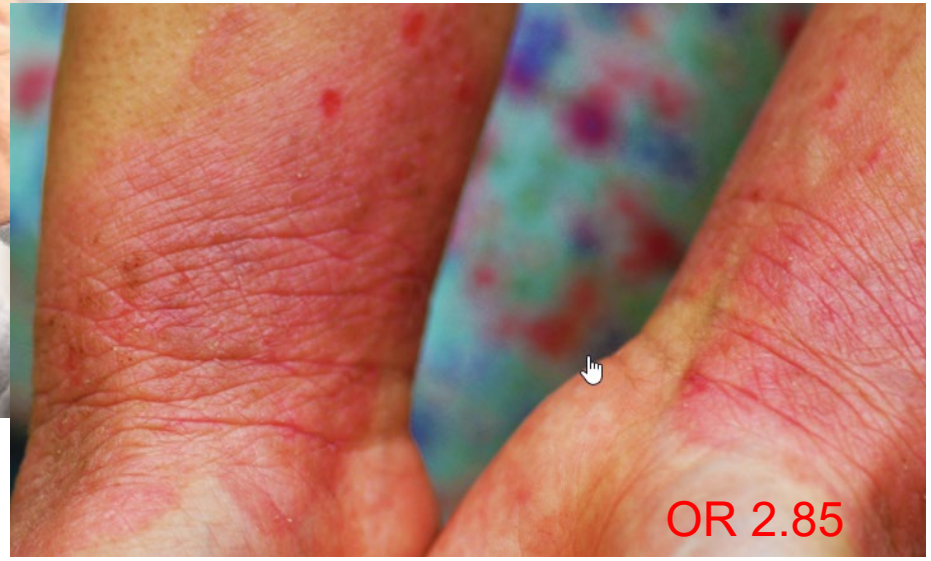
Mendelian disease associated with EoE	Inheritance	Genetic mutation	Plausible etiologic mechanism
Hyper-IgE syndrome	AD	Deleterious mutations in signal transducer and activator of transcription 3 (*STAT3*)	Dysregulated response to IL-6 and possibly IL-5
Hyper-IgE syndrome	AR	Loss-of-function mutations in dedicator of cytokinesis 8 (DOCK8)	Loss of T cell homeostasis; lack of durable secondary antibody response against specific antigens
Ehlers-Danlos syndrome, hypermobility type	AD	Unknown – other subtypes of Ehlers-Danlos syndrome are caused by mutations in collagen genes	Disrupted joint and skin development; increased activity of transforming growth factor beta (TGF-β) due to altered binding by extracellular matrix
ERBIN deficiency	AD	Loss-of-function mutation in ERBB2-interacting protein (ERBIN)	Increased TGF-β pathway activation in T cells with increased Th2 responses
Loeys-Dietz syndrome (LDS)	AR	Mutations in TGF-β receptors 1 and 2 (*TGFBR1* and *TGFBR2*, respectively)	Enhanced TGF-β signaling
Netherton syndrome	AR	Loss-of-function mutations in skin protease inhibitor, kazal type 5 (*SPINK5*)	Unrestricted protease activity of kallikrein 5 and 7 (KLK5, KLK7)
PTEN hamartoma tumor syndrome (PHTS)	AD	Mutations in phosphatase and tensin homolog (*PTEN*)	Inhibited regulation of the phosphatidylinositol-4,5-bisphosphate 3-kinase (PI3K) signaling pathway
Severe atopy syndrome associated with metabolic wasting (SAM) syndrome	AR	Homozygous mutations in desmoglein 1 (*DSG1*) or desmoplankin (*DSP*)	Disrupted epithelial barrier

AD, autosomal dominant; AR, autosomal recessive; EoE, eosinophilic esophagitis.

Risk factors

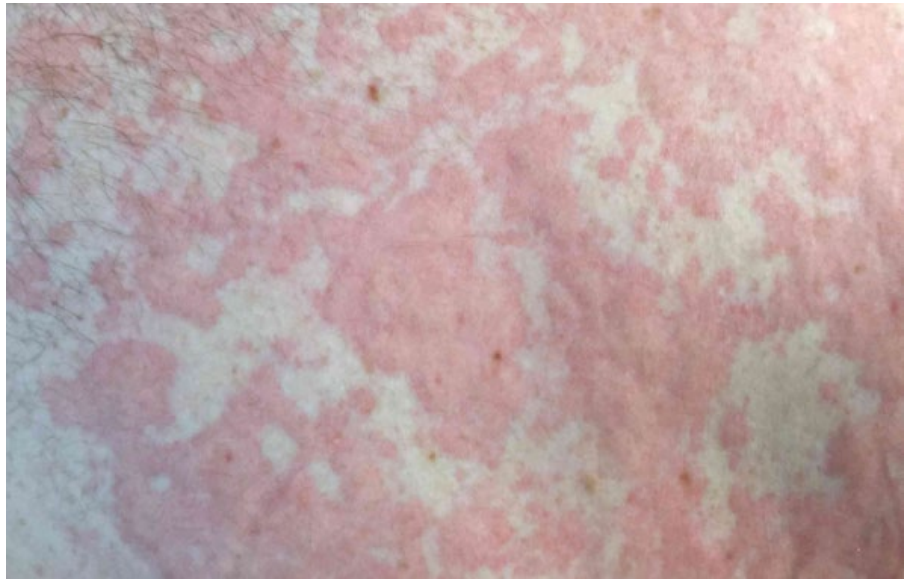
- **Atopy**

50-60% EoE patients are atopic and sensitized to food and aeroallergens



González-Cervera J, Ann Allergy Asthma Immunol, 2017 May;118(5):582-590

Risk factors



- Increased rate of anaphylaxis
  - 24% in cohort of EoE patients

Kugnanam KKN. Allergy. 2007 Nov

- Increased rate in patients with IgE-mediated food allergy (4.7% vs. 0.04% in normal population)

Hill DA. J Allergy Clin Immunol. 2017 Mar

- Oral immunotherapy for food allergies
  - Risk of 2.72% for de novo EoE

Lucendo AJ. Ann Allergy Asthma Immunol 2014

Potentially predictive factor of subsequent EoE

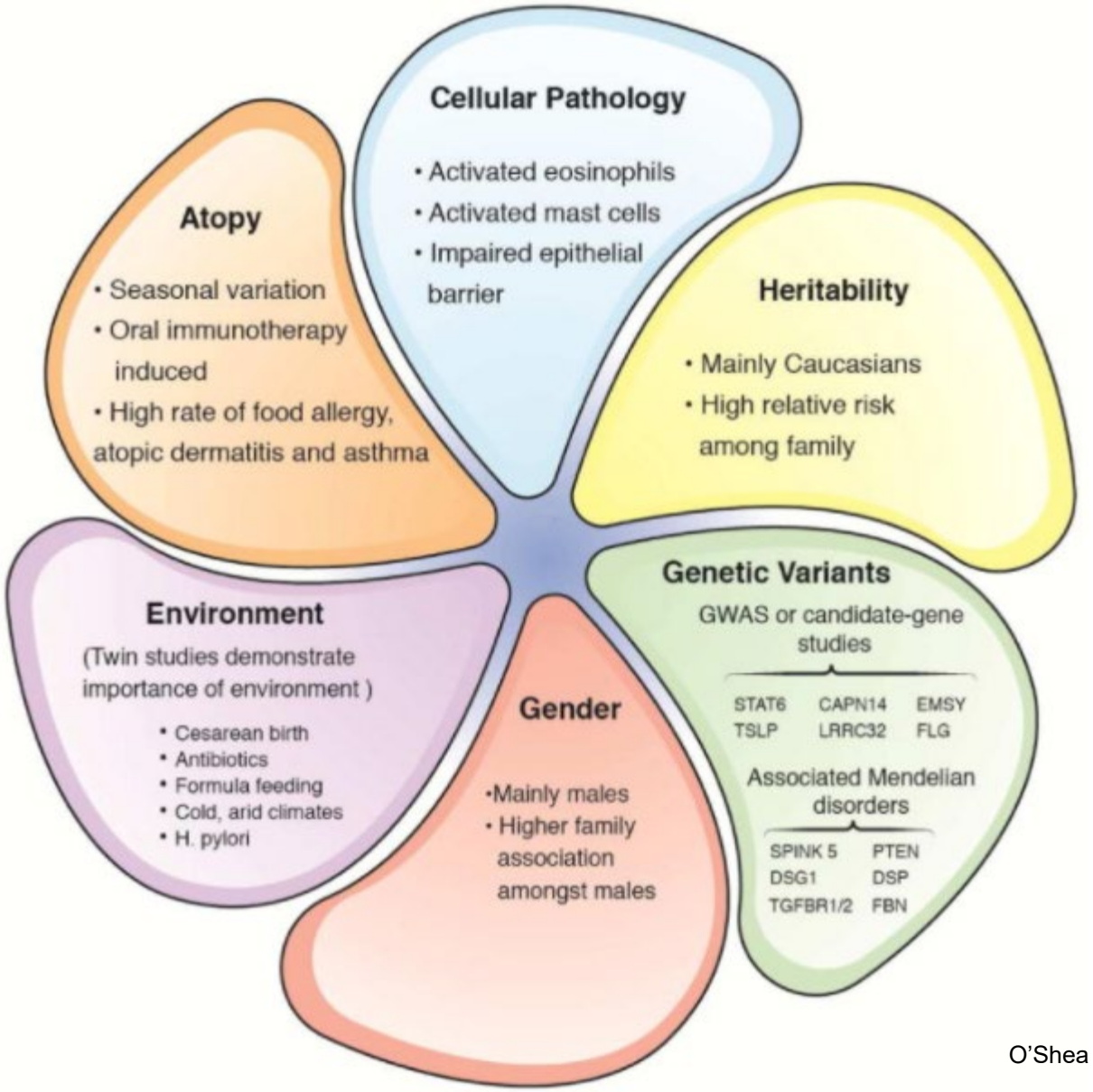
Further risk factors

- **Developmental/environmental**
  - C-section
  - Premature birth
  - Antibiotics in childhood
  - No breastfeeding
  - Cold and dry climate
  - H.pylori (Inversely proportional!)

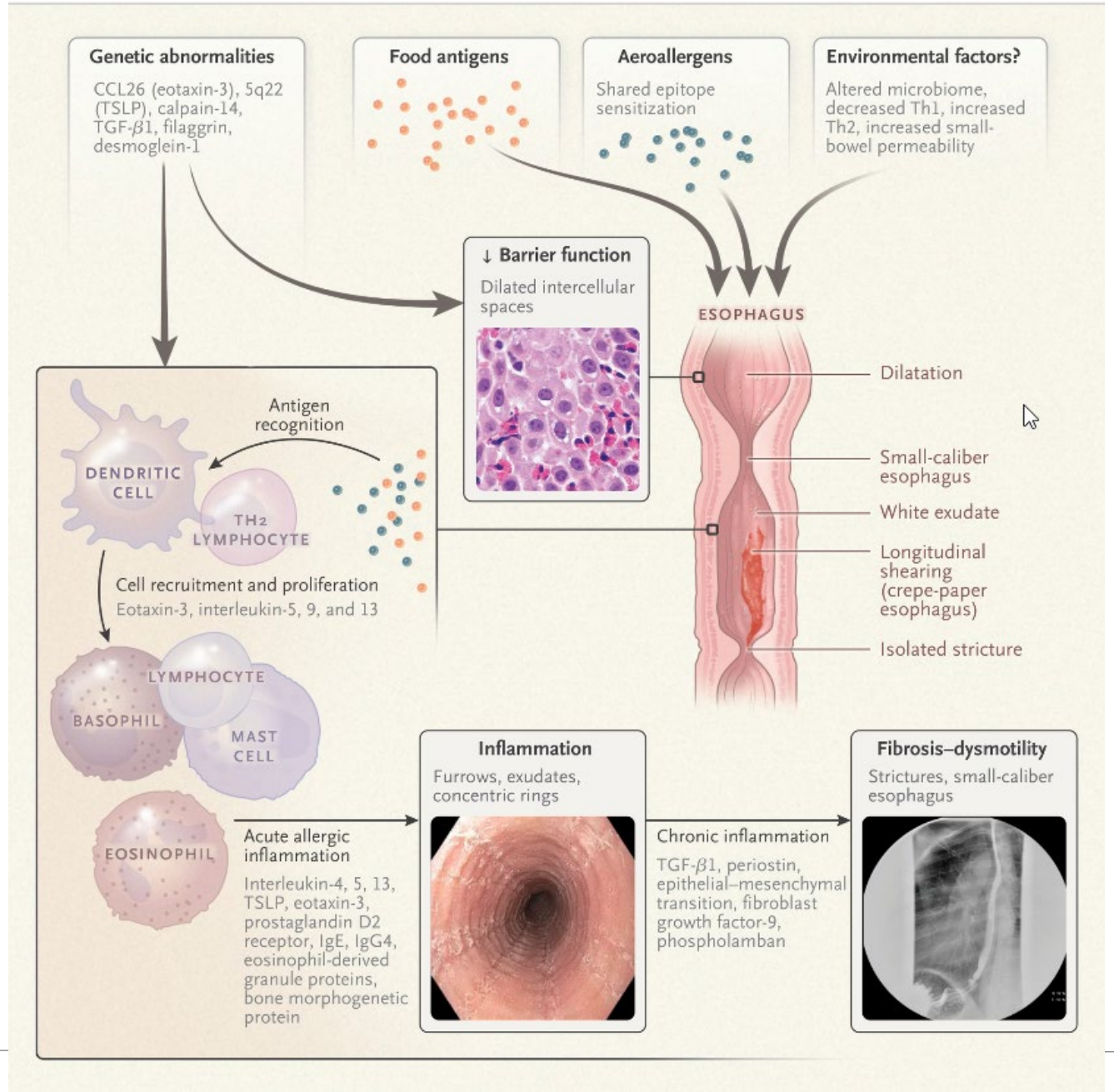
Modulation of microbiota

↑ Firmicutes and Proteobacteriae
↓ Streptococci

Skewing toward Th2-inflammation type
Similar to atopic diseases



O'Shea KM. Gastroenterology. 2018 Jan


Genetic abnormalities
CCL26 (eotaxin-3), 5q22 (TSLP), calpain-14, TGF-β1, filaggrin, desmoglein-1
Food antigens
Aeroallergens
Shared epitope sensitization
Environmental factors?
Altered microbiome, decreased Th1, increased Th2, increased small-bowel permeability
↓ Barrier function
Dilated intercellular spaces
ESOPHAGUS
Dilatation
Small-caliber esophagus
White exudate
Longitudinal shearing (crepe-paper esophagus)
Isolated stricture
Antigen recognition
DENDRITIC CELL
TH2 LYMPHOCYTE
Cell recruitment and proliferation
Eotaxin-3, interleukin-5, 9, and 13
LYMPHOCYTE
BASOPHIL
MAST CELL
EOSINOPHIL
Acute allergic inflammation
Interleukin-4, 5, 13, TSLP, eotaxin-3, prostaglandin D2 receptor, IgE, IgG4, eosinophil-derived granule proteins, bone morphogenetic protein
Inflammation
Furrows, exudates, concentric rings
Chronic inflammation
TGF-β1, periostin, epithelial–mesenchymal transition, fibroblast growth factor-9, phospholamban
Fibrosis–dysmotility
Strictures, small-caliber esophagus

Pathophysiology

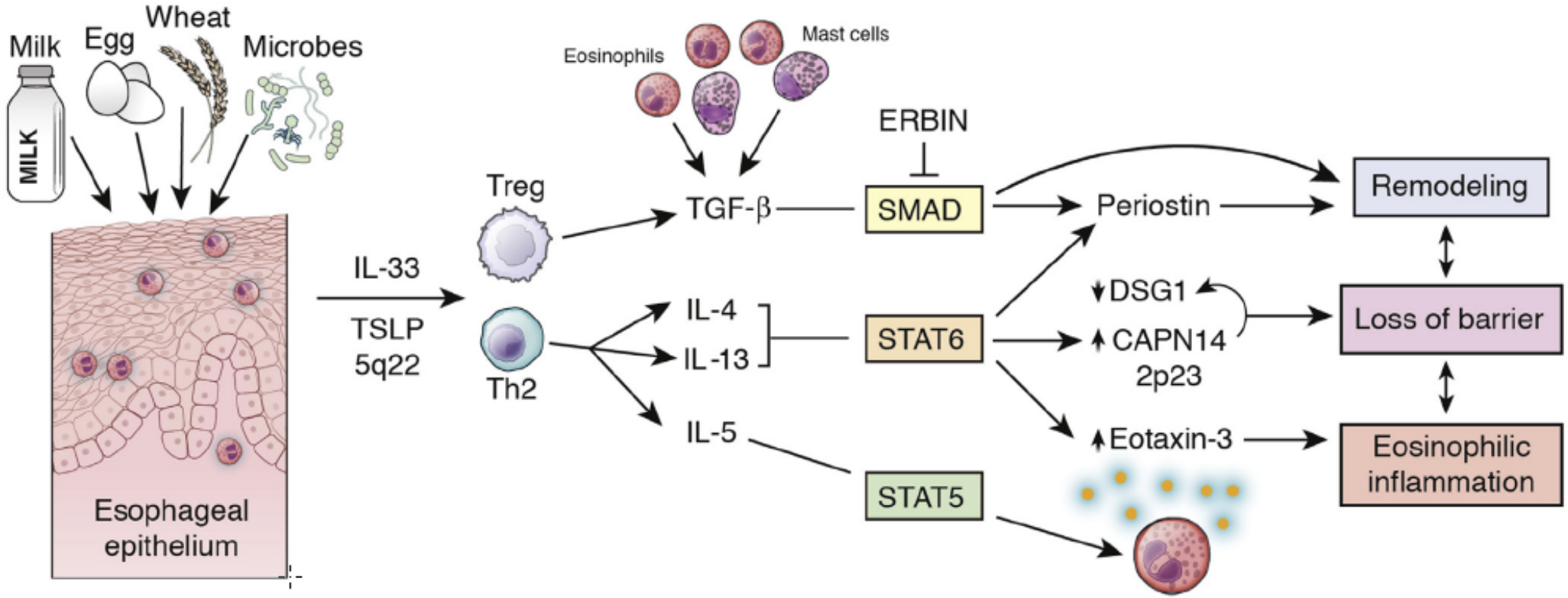
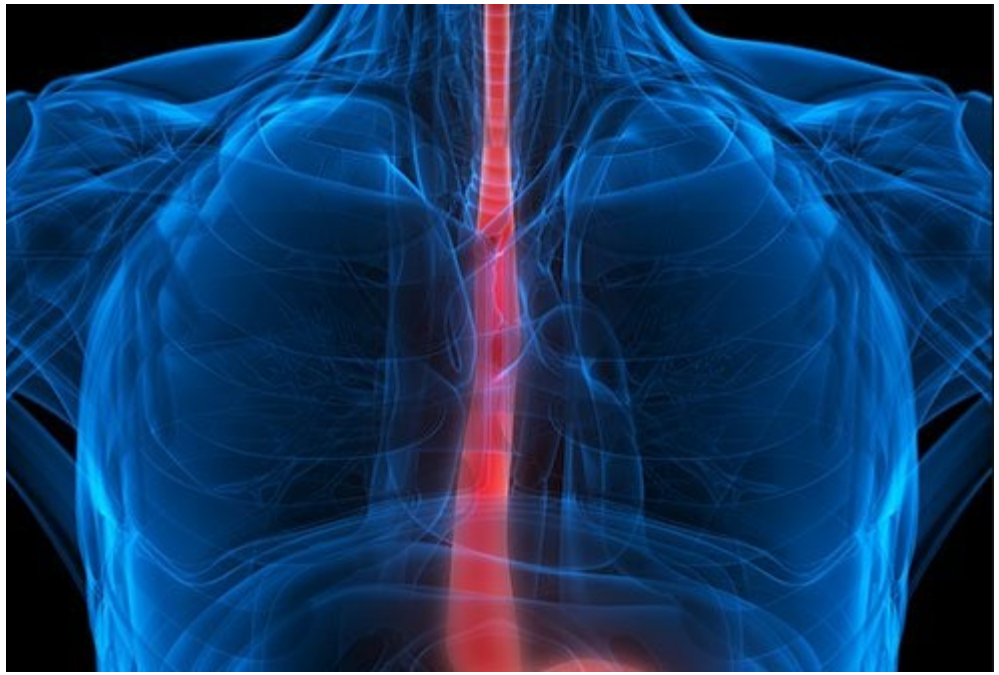


Figure 1. Pathophysiologic overview of EoE. Environmental factors, including foods and the microbiome, interact with the esophageal epithelium to elicit production of the proatopy cytokines IL-33 and TSLP. Activated T regulatory and T helper type 2 cells secrete bioactive cytokines including TGF-β, IL-4, IL-13, and IL-5, which elicit barrier disruption, tissue remodeling, and eosinophilic inflammation.

Symptoms



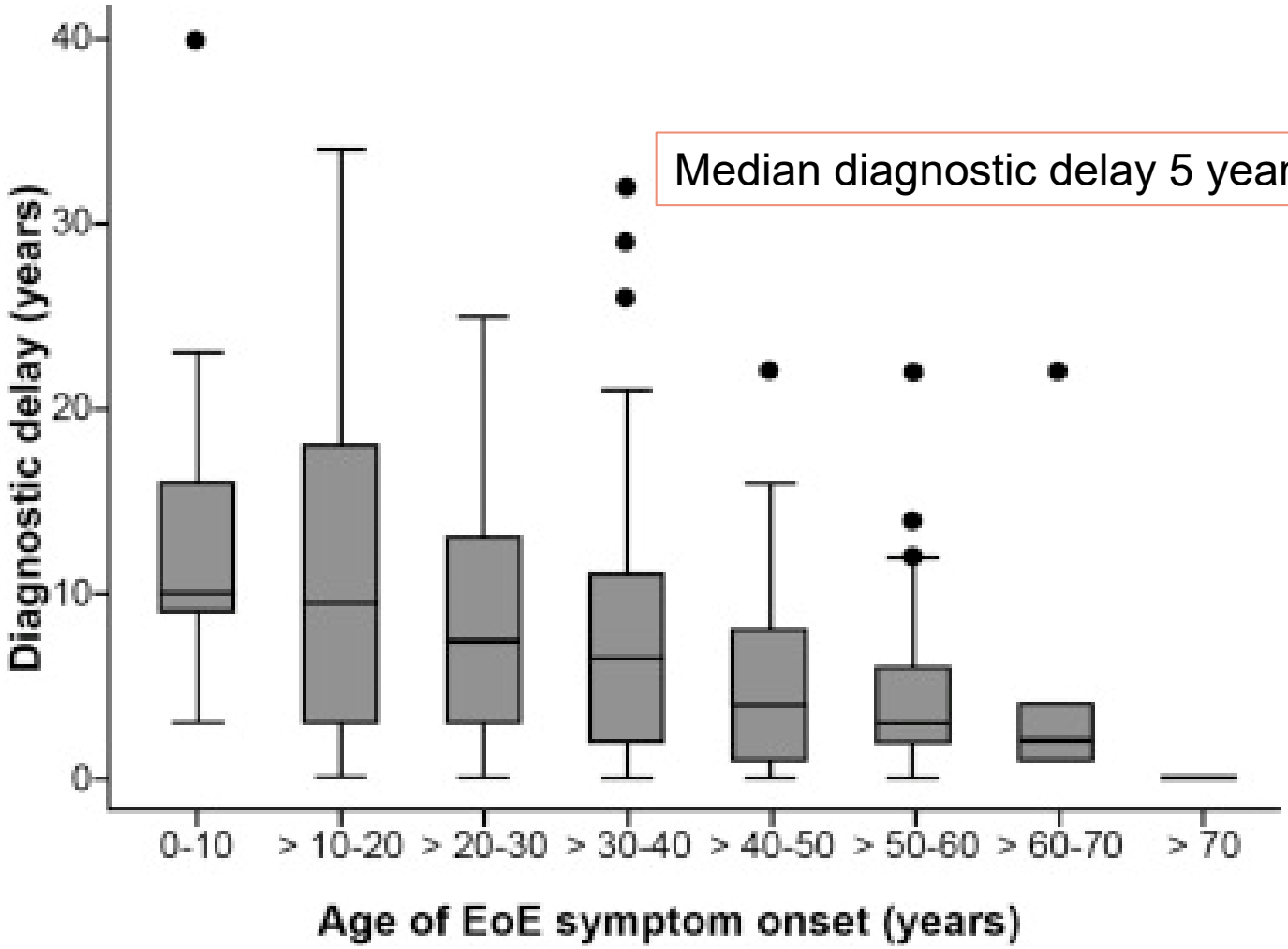
- Adults
  - Dysphagia (70-80%)
  - Impactions (33-54%)
  - Retrosternal pain or burning
  - Regurgitations
  - Often underestimated due to compensation mechanisms (slow eating/chewing, fluid intake, avoidance of eliciting food)
  - Symptom burden does not correlate to histological extent of inflammation

Symptoms

- Children
  - Reflux-like
  - Reduced growth
  - Vomiting
  - Abdominal pain
  - Avoidance of food

Symptoms

- Prevalence of EoE
  - Esophageal sy
  - Dysphagia
  - Impactions



Median diagnostic delay 5 years (IQR 2-10 years)

Schoepfer AM et al, Gastroenterology 2013;145:1230

Diagnosis

- Combination of symptoms, endoscopy, histology

- Endoscopy with biopsies
  - 6-8 biopsies, different locations, most importantly mucosal changes
  - Oral and aboral
  - Even when normal mucosa

- Biopsies stomach/duodenum to exclude EGID

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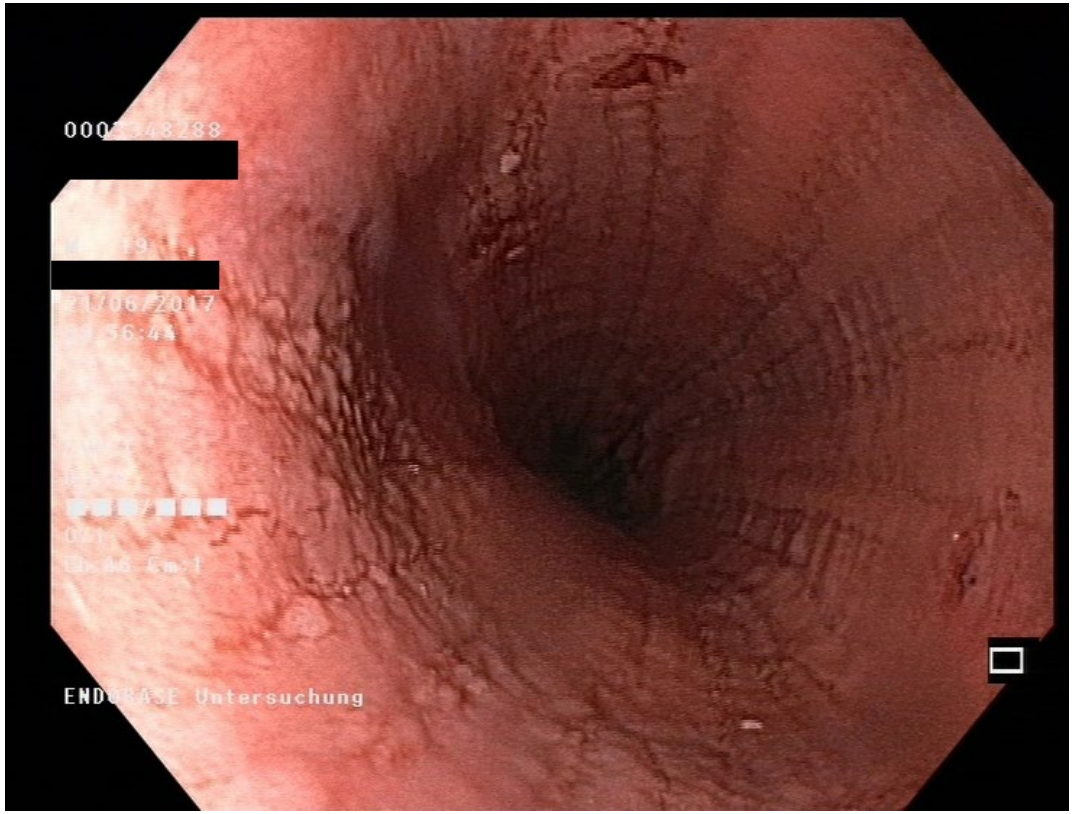
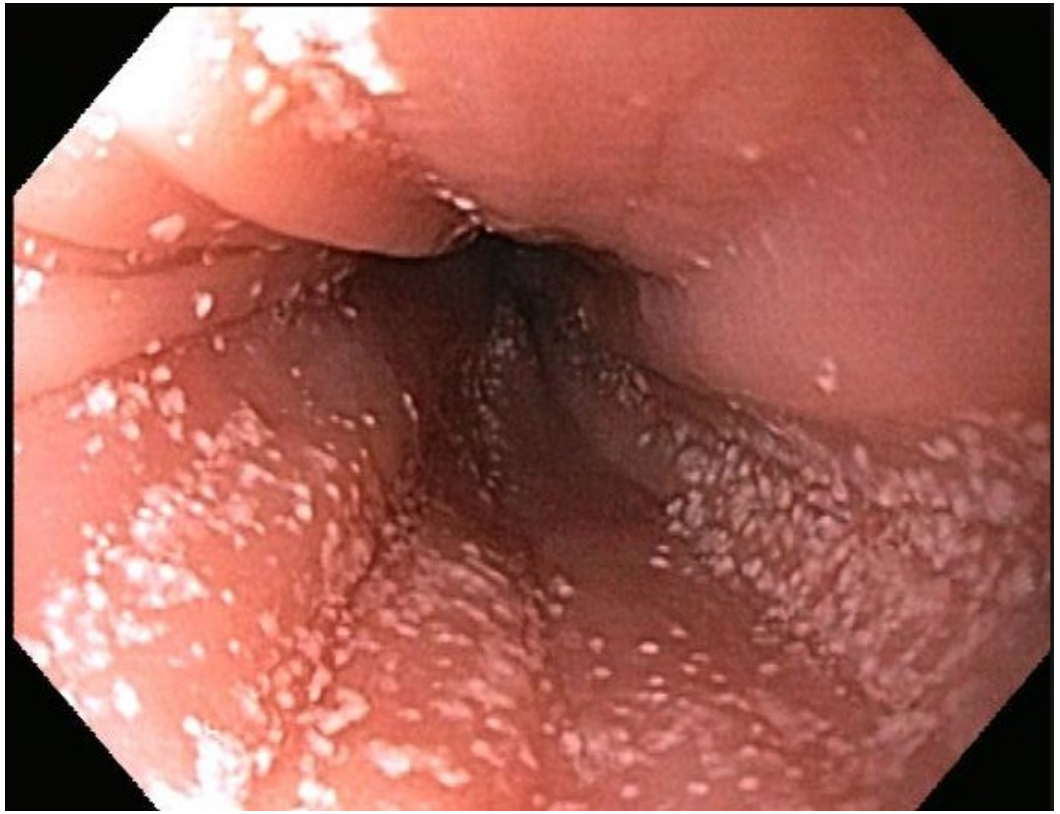
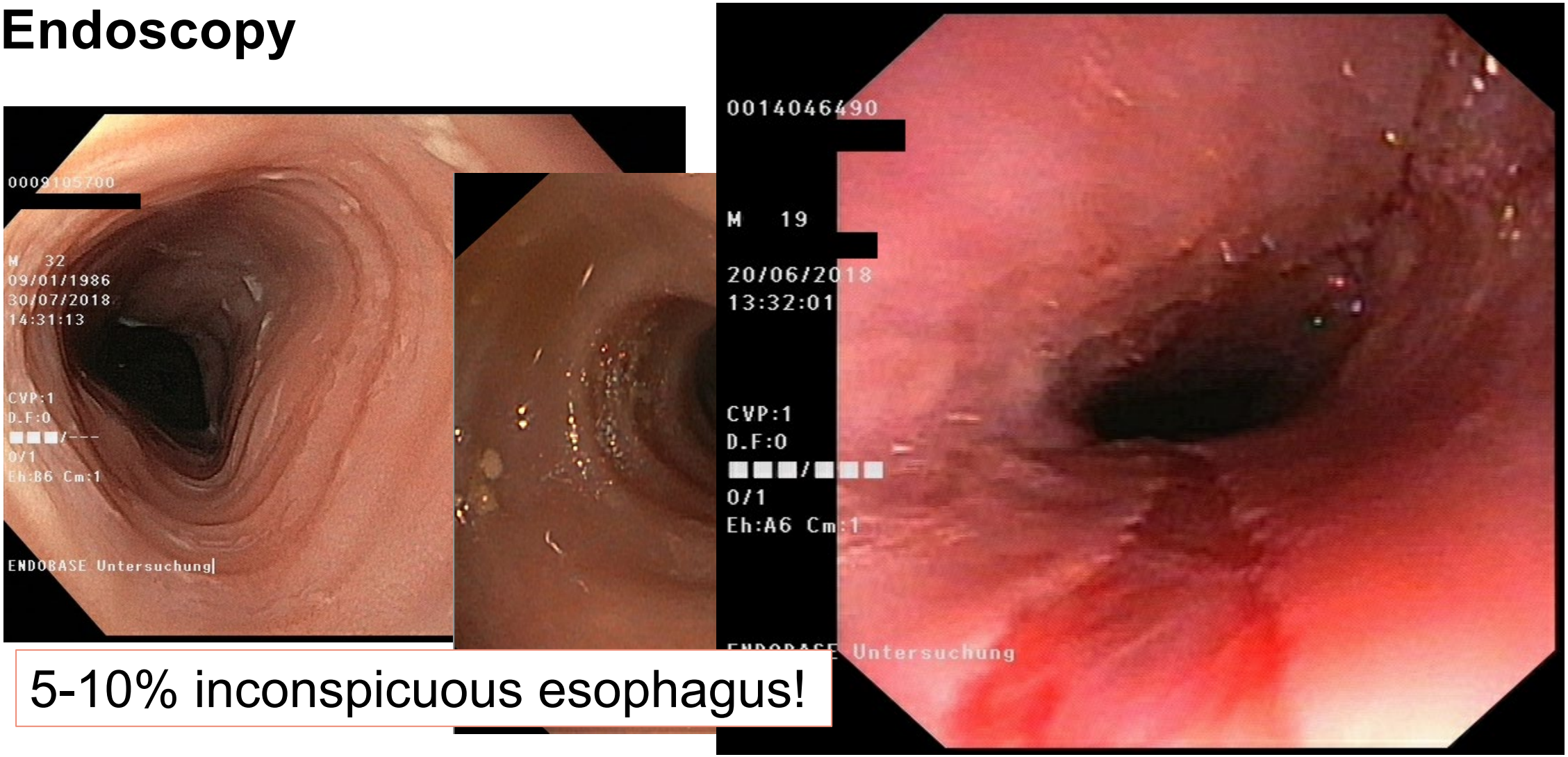
Endoscopy





Endoscopy



5-10% inconspicuous esophagus!

EREFS

Edema (loss vascular markings)

Grade 0: Distinct vascularity
Grade 1: Decreased
Grade 2: Absent

Rings (trachealization)

Grade 0: None
Grade 1: Mild (ridges)
Grade 2: Moderate (distinct rings)
Grade 3: Severe (not pass scope)

Exudate (white plaques)

Grade 0: None
Grade 1: Mild (≤10% surface area)
Grade 2: Severe (>10% surface area)

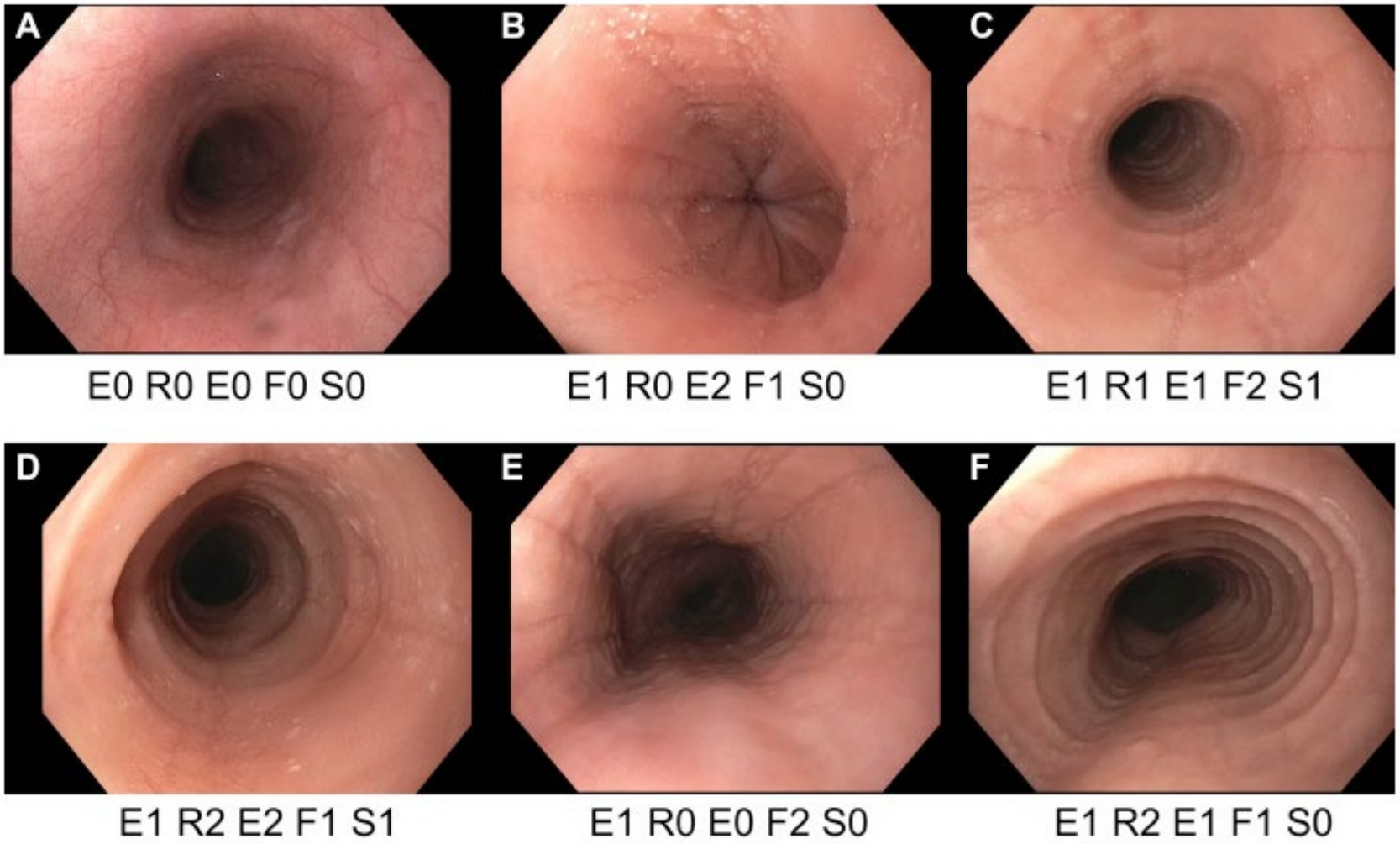
Furrows (vertical lines)

Grade 0: None
Grade 1: Mild
Grade 2: Severe (depth)

Stricture

Grade 0: Absent
Grade 1: Present

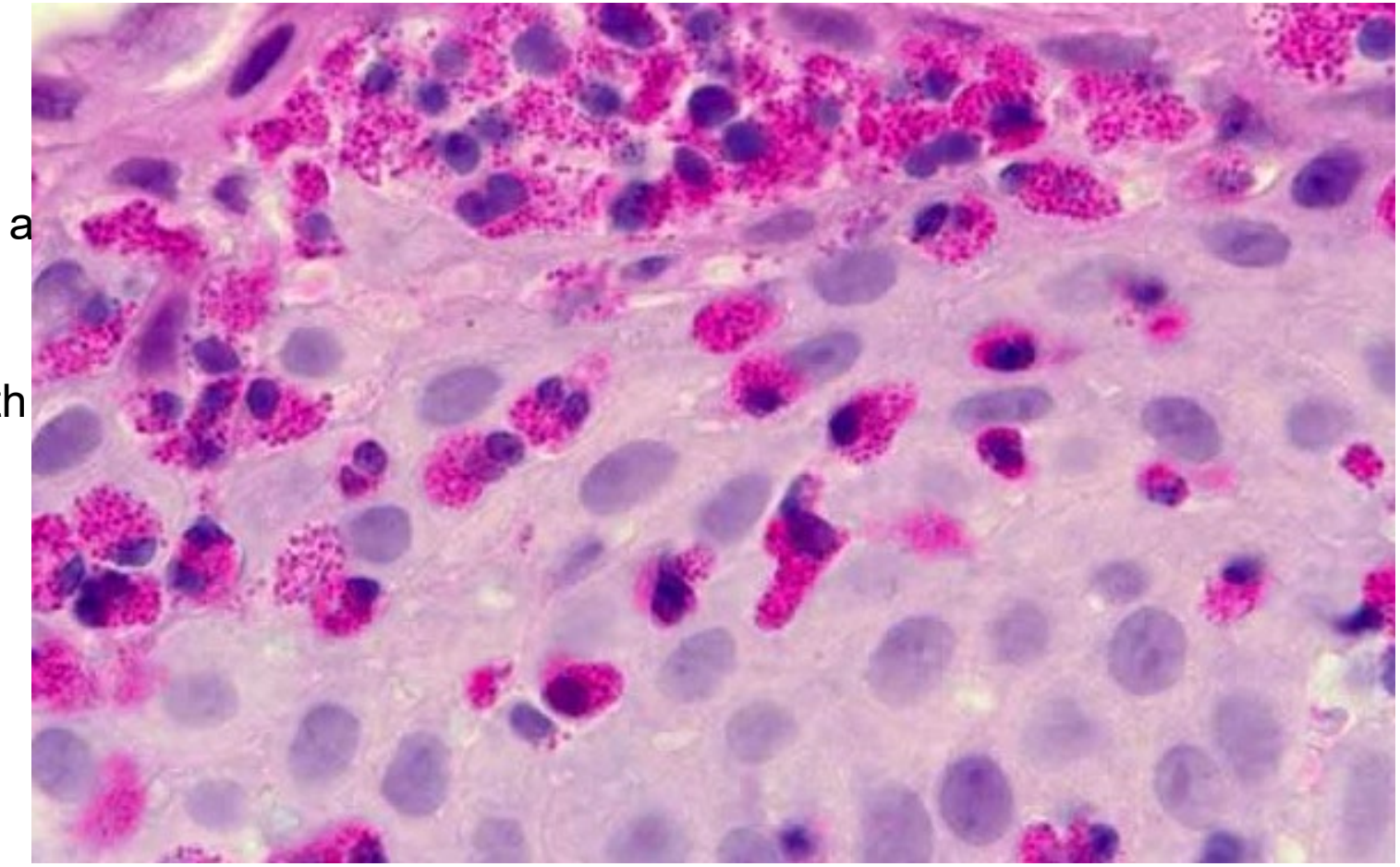
EREFS


A
B
C
E0 R0 E0 F0 S0
E1 R0 E2 F1 S0
E1 R1 E1 F2 S1
D
E
F
E1 R2 E2 F1 S1
E1 R0 E0 F2 S0
E1 R2 E1 F1 S0

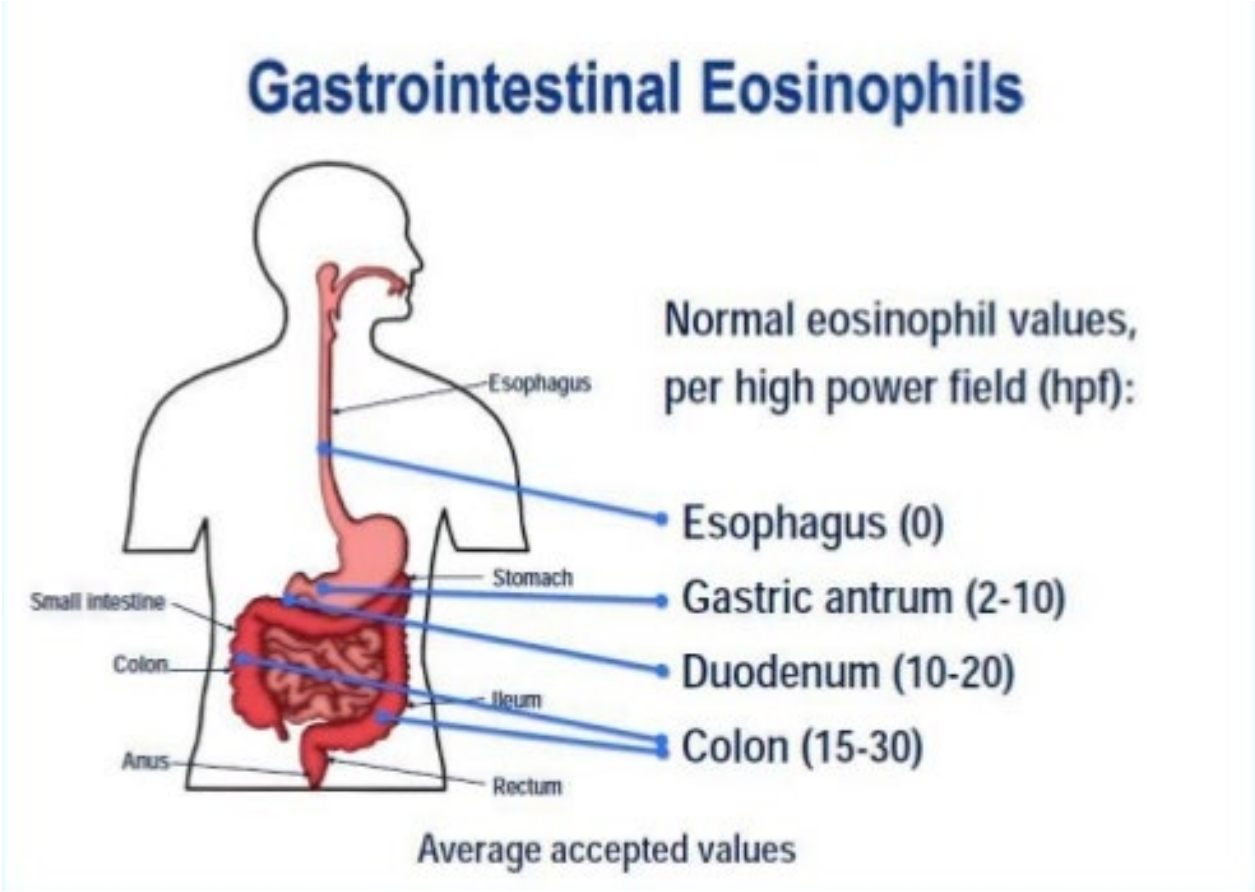
Histology

- Hämatoxylin a

- Histology with



Normal distribution of eosinophils in GIT



Differential diagnosis

- Esophageal eosinophilia
  - GERD, achalasia
  - Eosinophilic gastroenteritis (EGID)
  - Crohns disease
  - Medication (NSAID, Clozapin, Rifampicin, Enalapril, Tacrolimus, Carbamazepin)
  - Infections (Protozoa, parasites)
  - Celiac disease
  - Hypereosinophilic syndrome
  - Rarely: collagenoses, vasculitides, pemphigus, GvHD, lymphoproliferative diseases

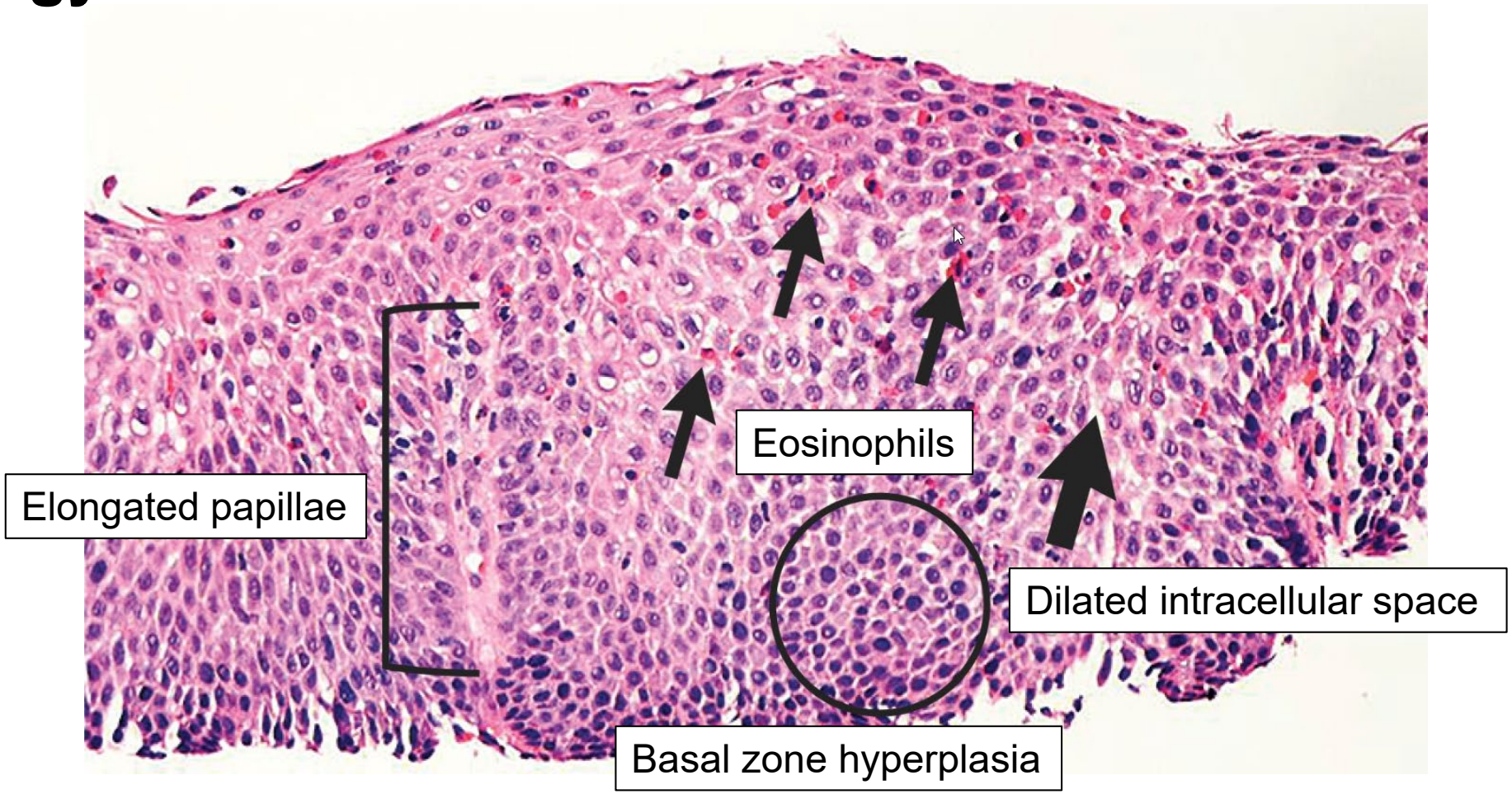
EoE and GERD

- Overlapping symptoms, often different patient characteristic
- Co-existing with or without effect, but may interact bidirectionally

- Interactions
  - Reflux induced increase in permeability
  - Altered motiliy in EoE inhibiting acid clearance
  - Acid-hypersensitivity in EoE

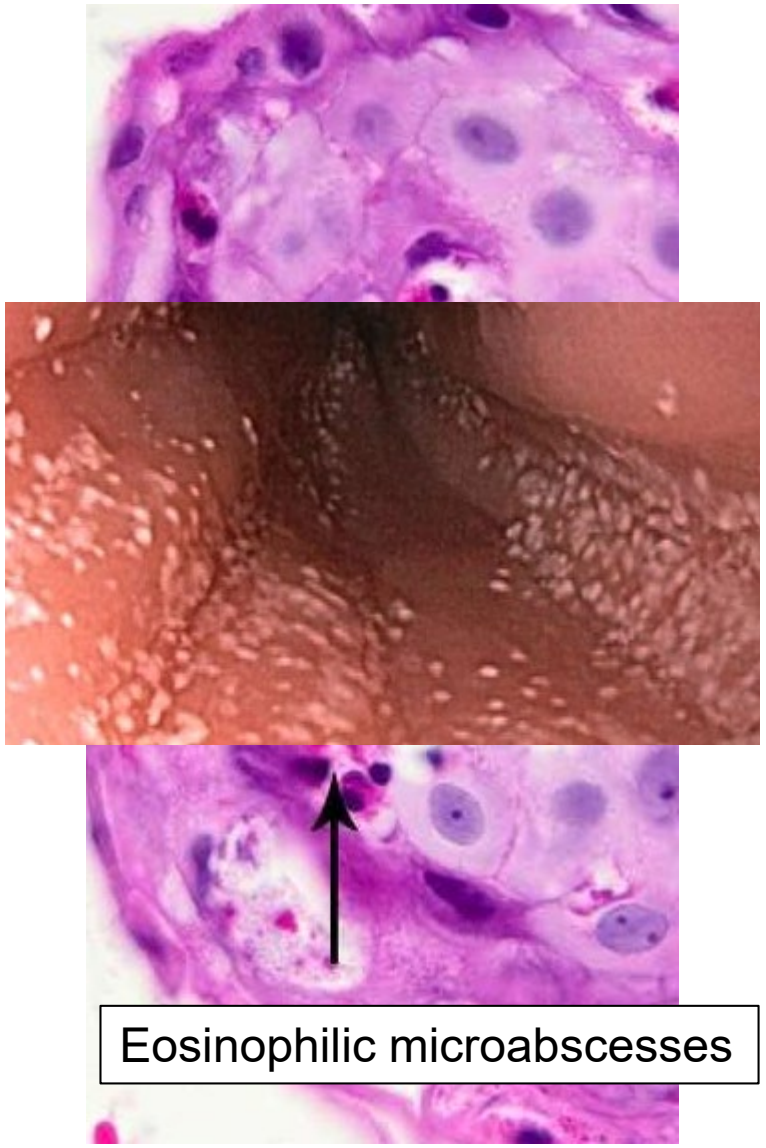
- Differentiation with 24h-impedance-pH-metry

Histology


Elongated papillae
Eosinophils
Dilated intracellular space
Basal zone hyperplasia

Histology

EoE>GERD



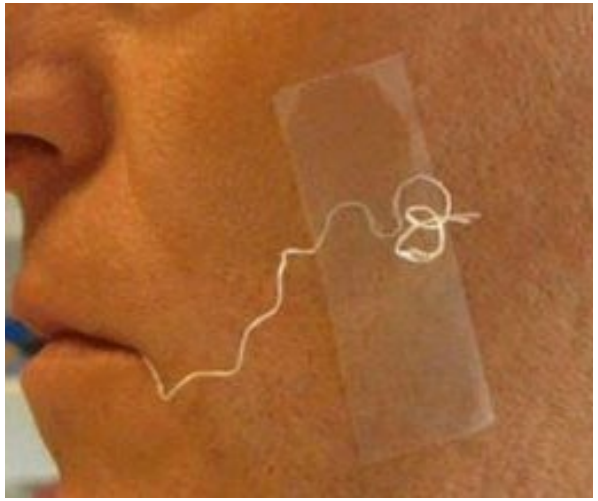
Non-invasive markers

- Cytosponge
    - 75% sensitivity, 86% specificity, good correlation sponge/endoscopy

Katzka D. Am J Gastroenterol. 2017 Oct;112(10):1538-1544

- String test
    - Measuring eotaxin-3 and major basic protein-1 concentrations (AUC 0.86)

Ackermann S. Am J Gastroenterol. 2019 Oct



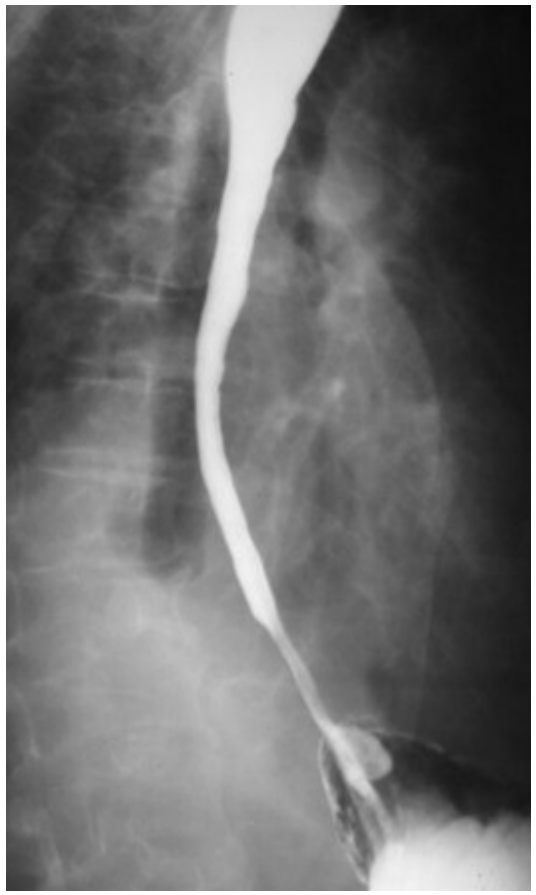
Radiology/barium swallow

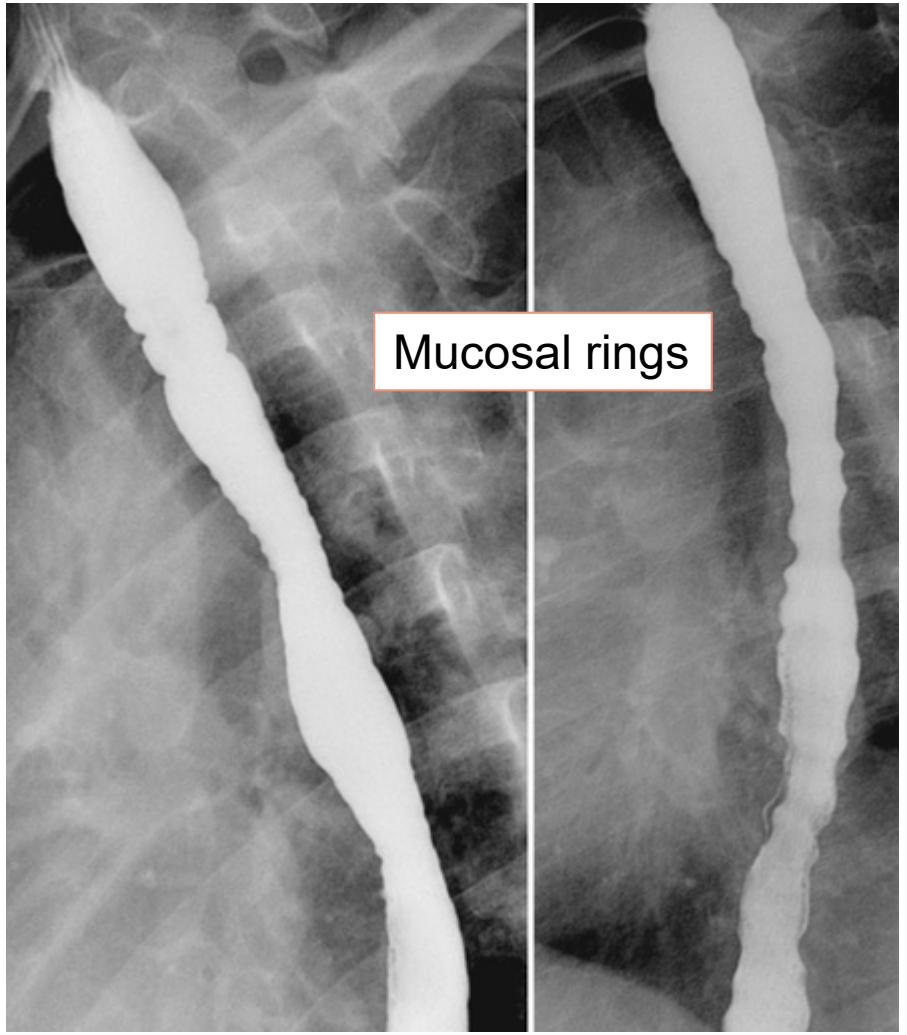
- Not helpful in diagnostics

- BUT

  -> Important for stenoses

  - Amount
  - Location
  - Extension and diameter

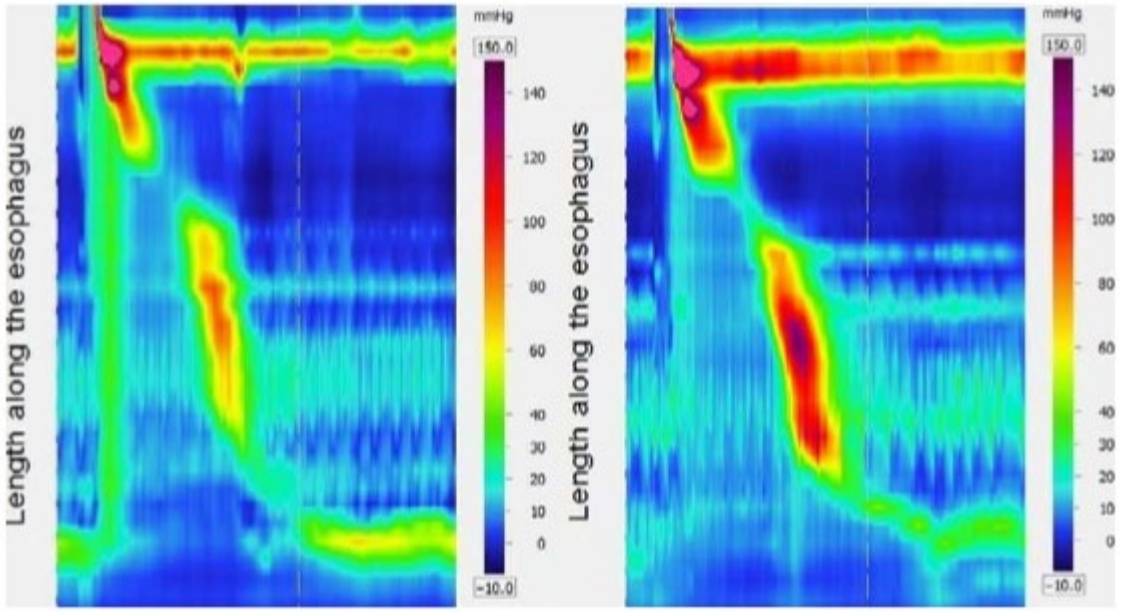




HR-manometry

- 1/3 with altered motility[1,2]
- 86% improve after therapy[1]

- Frequent findings[1,2]
  - Early, panesophageal increase in pressure
  - Weak and insufficient peristalsis



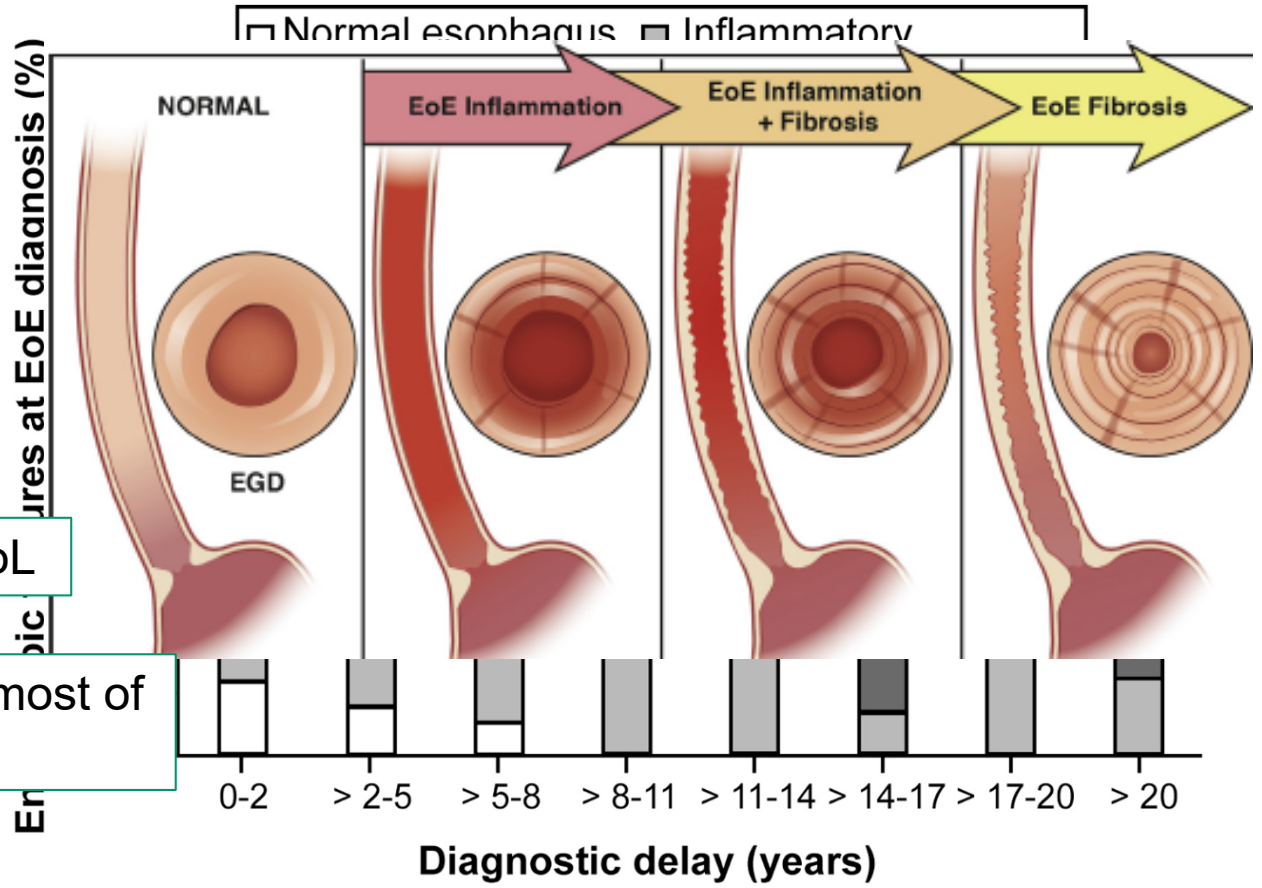
[1]Nennstiel S et al. High-resolution manometry in patients with eosinophilic esophagitis under topical steroid therapy—a prospective observational study (HIMEOS-study). Neurogastroenterol Motil (2016) 28, 599–607
[2]Roman S et al. Manometric features of Eosinophilic Esophagitis in Esophageal Pressure Topography. Neurogastroenterol Motil. 2011 Mar; 23(3): 208–e111.

Natural course

- Chronic progressive inflammation
- Fibrotic tissue remodelling
- Strictures

Decreas in QoL

No predisposition of other diseases, most of all cancer



Schoepfer AM et al, Gastroenterology 2013;145:1230

Complications

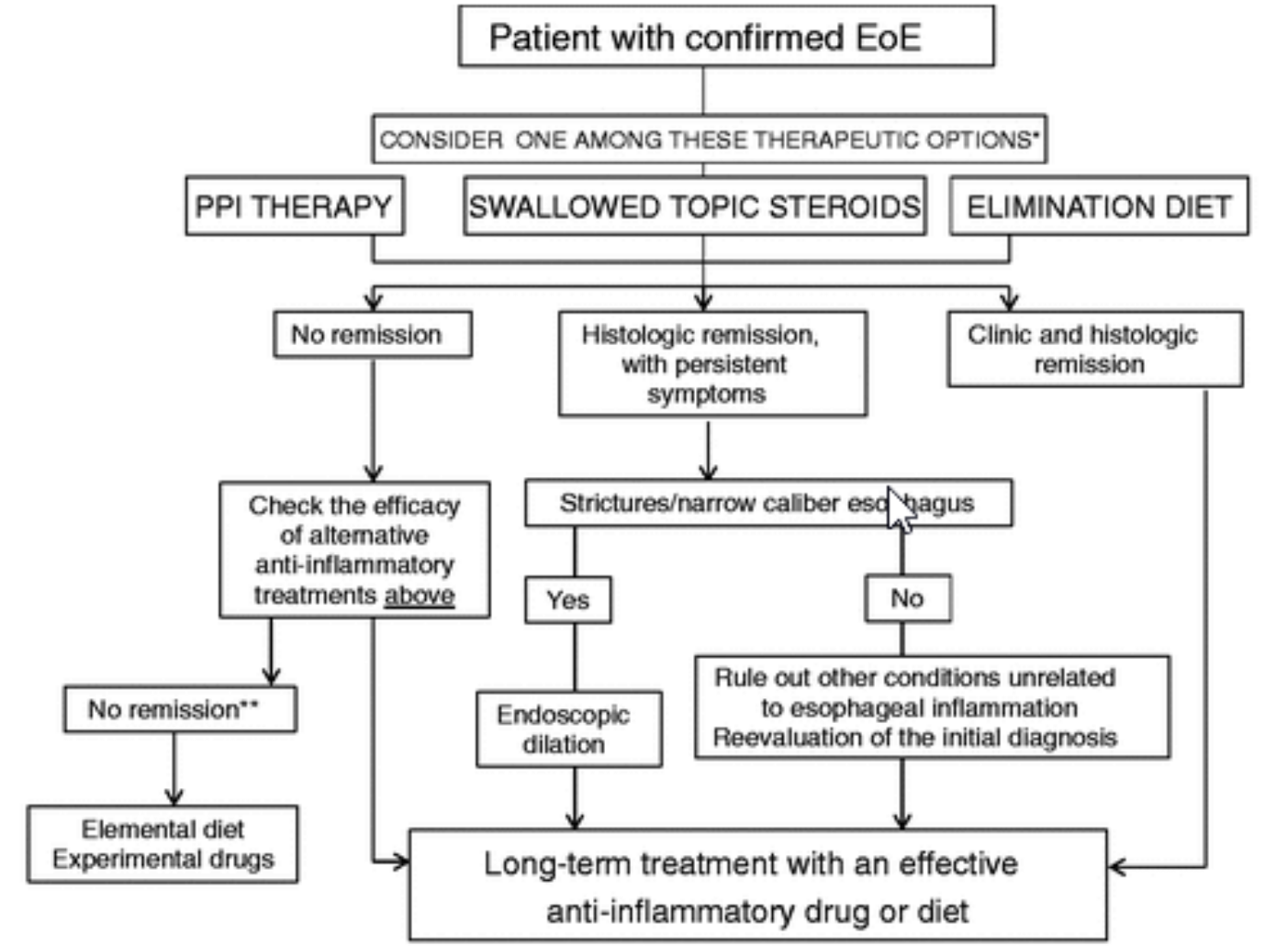
- Strictures
- Food impaction
- Boerhaave
- Iatrogenic perforation
- Malnutrition
- No association to neoplasm, IBD, celiac disease or hypereosinophilia

Dilatation

- Treatment of stenoses
  - Esophageal diameter <13mm, strictures
  - Mostly after treatment
  - Rarely first line -> impactations and daily dysphagia
  - No substitute of medical therapy

  - Significantly reducing dysphagia in approx. 75%, lasting up to 1 year
  - No increased risk for perforation (<1%)

Treatment



*In patients with persistent symptoms under anti-inflammatory therapy, endoscopic dilation should be considered

** Refer the patient to an EoE center

PPI

- Potential therapeutic effect in some patients
- 2xdie > 1x/die
- Maintenance of remission possible (70% after 1y)
- Recurrence of eosinophils 3-6 months after treatment stopp

Gutiérrez-Junquera C. J Pediatr Gastroenterol Nutr. 2018 Aug
Molina-Infante J. Am J Gastroenterol. 2015 Nov

Supplementary Table 3. Evidence for Statements 23 (Effectiveness of proton pump inhibitor drugs for induction of histological remission EoE patients.

	Quality assessment					Summary of findings				
Studies	Risk of bias	Inconsistency	Indirectness	Imprecision	Other considerations[a]	Quality of evidence	Effectiveness rate	95% CI	Comparator	Comments
Proton pump inhibitor drugs for achieving histologic remission in EoE (importance of outcome: critical for decision making) Efficacy: Proportion of patients with <15 eos/hpf after therapy.										
1 SR (25 observational studies & 2 RCT)[215]	moderate[b]	moderate	none	none	Different drug, doses and duration	⊕⊕⊕∅ Moderate	50.46 %	42.2 – 58.71	NA	17 studies included adult patients; 11 included pediatric patients
Symptomatic improvement after proton pump inhibitor drugs (importance of outcome: critical for deci										
1 SR (24 observational studies & 1 RCT)[215]	moderate[b]	high	none	moderate	Different drug, doses and duration.	⊕∅∅∅ Very Low	60.8 %	48.38 – 72.2	NA	15 studies included adult patients; 11 included pediatric patients. No validated instruments were used to assess symptoms

If Eos <5 defined, then 33%

[a]Including publication bias.
[b]Mainly due to lack of blinding.

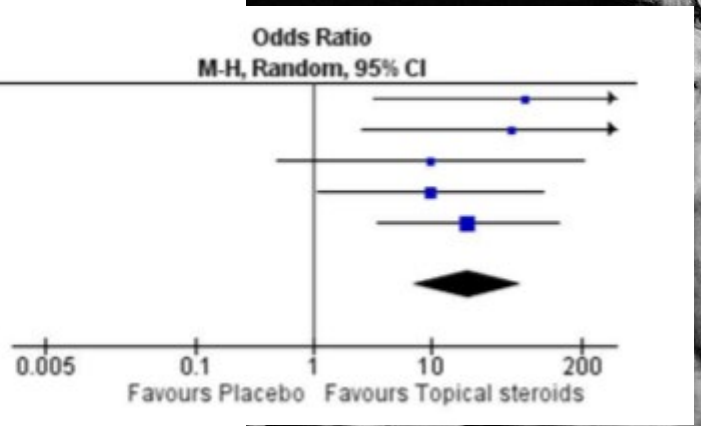
Lucendo AJ. Efficacy of proton pump inhibitor drugs for inducing clinical and histologic remission in patients with symptomatic esophageal eosinophilia: a systematic review and meta-analysis. Clin Gastroenterol Hepatol 2016;14:13–22

Steroids

- Highly effective in induction and maintenance of remission
  -
- Bu

Study or Subgroup	Topical steroids Events	Total	Placebo Events	Total	Weight	Odds Ratio M-H, Random, 95% CI
Alexander 2012	13	19	0	15	13.4%	64.38 [3.31, 1251.90]
Butz 2014	15	23	0	13	13.6%	49.24 [2.59, 935.08]
Dohil 2010	5	15	0	9	12.9%	9.95 [0.48, 204.99]
Konikoff 2006	10	20	1	11	23.6%	10.00 [1.07, 93.44]
Straumann 2010	13	18	2	18	36.5%	20.80 [3.45, 125.30]
Total (95% CI)		**95**		**66**	**100.0%**	**20.81 [7.03, 61.63]**
Total events	56		3			

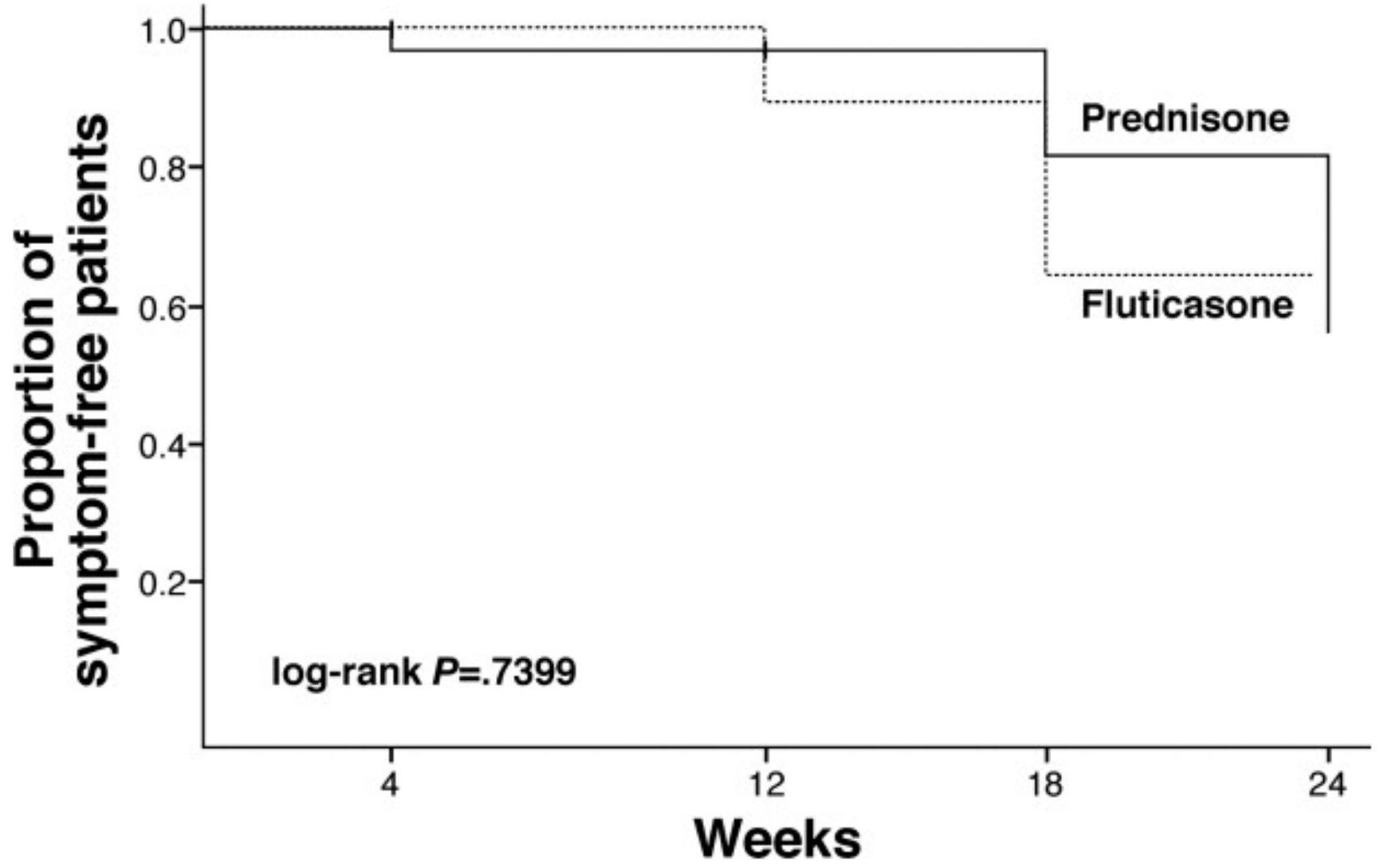
Heterogeneity: Tau² = 0.00; Chi² = 1.54, df = 4 (*P* = 0.82); I² = 0%
Test for overall effect: Z = 5.48 (*P* < 0.00001)



- Systemic steroids without relevant benefit!
  - Same effect, more adverse events (40%!)
  - Effect in refractory disease not known

Clin Gastroenterol Hepatol 2008;6:165–173

Stero


Proportion of symptom-free patients
1.0
0.8
0.6
0.4
0.2
Prednisone
Fluticasone
log-rank P=.7399
4
12
18
24
Weeks

Steroids

- I
- I

	NEB (n = 11)	OVB (n = 11)	p value
Primary outcomes			
Overall eosinophil counts (eos/hpf ± SD)			
Baseline maximum eosinophil count	101 ± 85	83 ± 89	0.62
Baseline mean eosinophil count	23 ± 20	20 ± 24	0.80
Post-treatment max eosinophil count*	89 ± 94	11 ± 23	0.02
Post-treatment mean eosinophil count*	31 ± 37	3 ± 7	0.02
Maximum eosinophil counts by level (eos/hpf)			
Baseline proximal esophagus	79 ± 73	54 ± 74	0.43
Post-treatment proximal esophagus†	57 ± 78	5 ± 17	0.04
Baseline mid esophagus	41 ± 47	59 ± 98	0.62
Post-treatment mid esophagus‡	55 ± 57	8 ± 22	0.02
Baseline distal esophagus	54 ± 66	53 ± 49	0.96
Post-treatment distal esophagus#	69 ± 81	11 ± 23	0.03

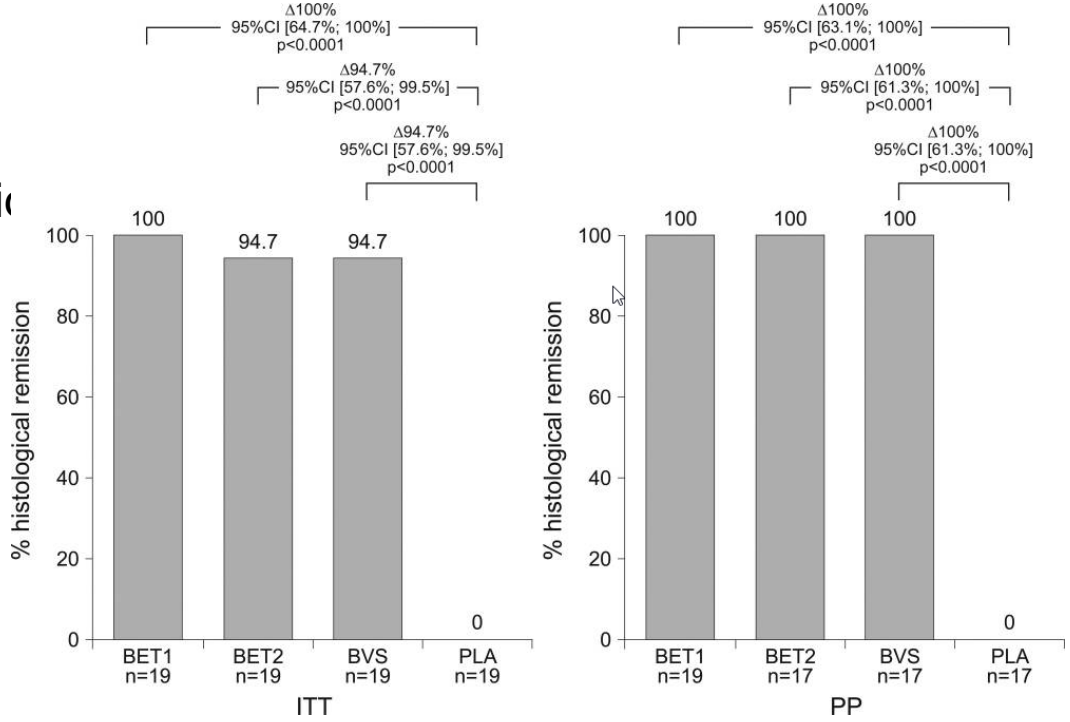
Secondary outcomes			
Mucosal medication contact time (median)			
Overall esophageal area under the curve	19200	48900	0.005
Proximal esophageal AUC	7300	14400	0.14
Mid esophageal AUC	2800	7800	0.01
Distal esophageal AUC	3800	18100	0.001
AUC with a complete histologic response	61000	65000	0.76
AUC without a complete response††	19200	34000	0.06

Maintenance of remission in ca. 60%

Dellon ES et al. Viscous topical is more effective than nebulized steroid therapy for patients with eosinophilic esophagitis. Gastroenterology 2012;143:321–324.

Steroids

- New: Jorveza
  - Orodispersible effervescent pill with budesonid
  - More pleasant than suspension
  - Single licensed therapy for this indication



Gut. 2016 Mar;65(3):390-9

Steroids

- No relevant adverse events
  - Budesonide: First pass effect in liver 90%

- Esophageal candidiasis in 10% (mostly incidental finding on endoscopy)
  - Almost exclusively asymptomatic
  - Unproblematic treatment (Nystatin, Fluconazol)

- In children potentially suprarenal insufficiency
  - Possibly monitoring of cortisol axis in patients with high dosis over long time

Nutrition

- Elemental diet
  - Only after therapy beforehand
  - Response rate 90% (children and adu
  - Clinical improvement in 8.5+
  - Histological remis
- BUT
  - Not really palatable (cooling, straw), sufficient compliance only in 1/3
  - QOL, social isolation
  - Costs (also endoscopies)

Arias A, Gonzalez-Cervera J, Tenias JM, et al. Efficacy of dietary interventions for inducing histologic remission in patients with eosinophilic esophagitis: a systematic review and meta-analysis. Gastroenterology 2014;146:1639–1648

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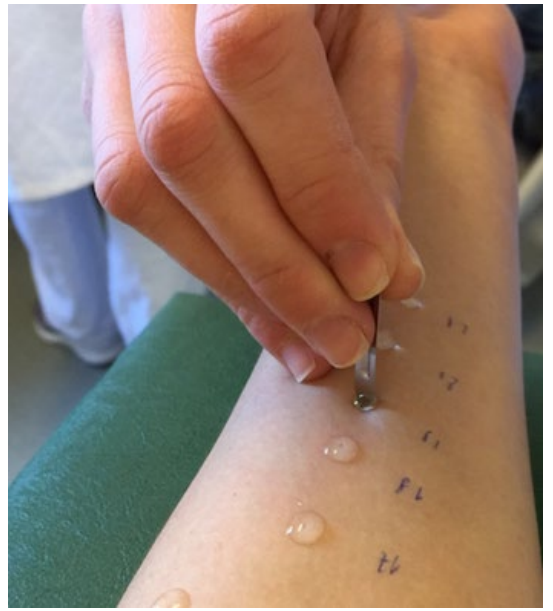
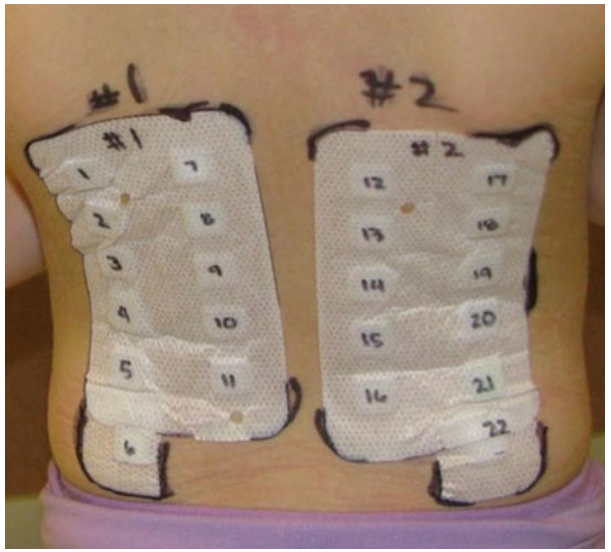
Nutrition and allergy



- Skin prick test (SPT), IgE-mediated reaction
  - PPV 47%
  - NPV >90% (CAVE milk 30%, egg/soy/wheat 79-90%)



- Atopy patch test (APT), non-IgE mediated reaction
  - PPV 44%
  - NPV >90% (milk 31%)

Nutrition and allergy

- Combination of both tests
  - Sensitivity 65–95% (milk/pork 50%); Specificity 78–90%
  - PPV 44%, NPV 92% (milk 40-44%, egg 56%, wheat 67%)

Spergel J. The use of skin prick tests and patch tests to identify causative foods in eosinophilic esophagitis. *J Allergy Clin Immunol. 2002 Feb; 109(2):363-8*

- Serum IgE-elimination diet (sIgE-ED)
  - Non-inferiority to SFED with histological response in 73%
  - Most eliminated foods were wheat (85%), nuts (73%) and cow's milk (61%)
  - Sensitivity 87.5%, specificity 68%. APT all negative

Rodriguez-Sanchez J. Efficacy of IgE-targeted vs empiric six-food elimination diets for adult eosinophilic oesophagitis. *Allergy.* 2014

Nutrition

- «Six food elimination diet»
  - First introduced in 2006
  - Overcoming the insufficent sens/spec of allergy testing
  - More realistic in real life than elemental diet

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Six food elimination diet

Nutrition

- Six food:
  - Remission rate of 72%!
- Four food: cow’s milk, wheat, eggs, soy/legumes
  - Remission 50%
- Two food: cow’s milk and wheat
  - Remission 40%

Efficient in maintenance of remission

- Problem: Control endoscopies after introduction of new agent
- Step-up approach reduces endoscopies

Combination?

- Potentially additive effect

- 4-FED + PPI vs. PPI mono showed higher rates of partial remission (<10 eos) after 12 weeks
  - 88% vs. 45%

Heine et al, J Allergy Clin Immunol. 2019

Other possible medication

- Azathioprine/6-MP
  - Potentially effective for maintenance of remission (steroid sparing), to little data

- Anti-allergic medication
  - Montelukast withouth enough evidence
  - Cromoglycine und anti-histamines without effect
  - CRTH2 antagonist (Fevipripant) with moderate improvement of symptoms and histology

Straumann A. Anti-eosinophil activity and clinical efficacy of the CRTH2 antagonist OC000459 in eosinophilic esophagitis. *Allergy.* 2013

Biologics

- Anti-IgE antibody (Omalizumab)
  - No effect

    Clayton F, Gastroenterology, 2014

- Anti-TNFα Antikörper (Infliximab)
  - No effect

    Straumann A. J Allergy Clin Immunol. 2008 Aug

Biologics

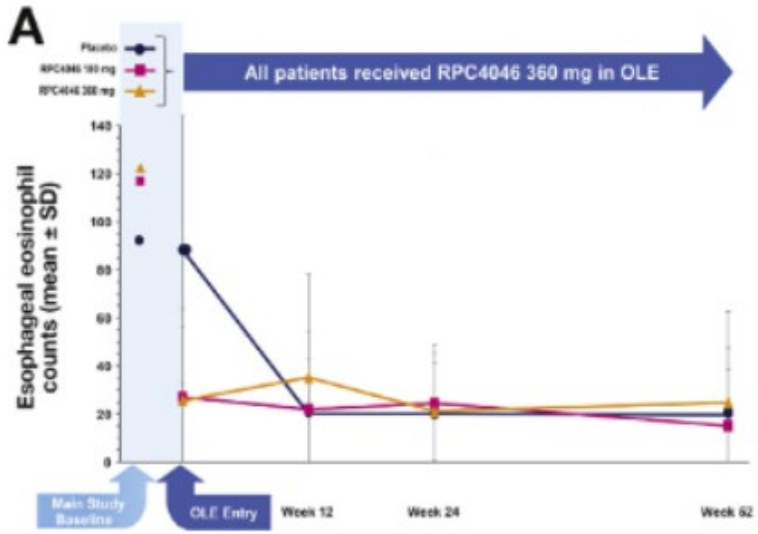
- Anti-IL 5 antibody (Mepolizumab and Reslizumab) Straumann A et al. *Gut.* 2010 and Spergel J.M. et al. *J Allergy Clin Immunol.* 2012
  - No improvement of symptoms
  - Significant reduction of eosinophilia (50-60%), but no histological remission

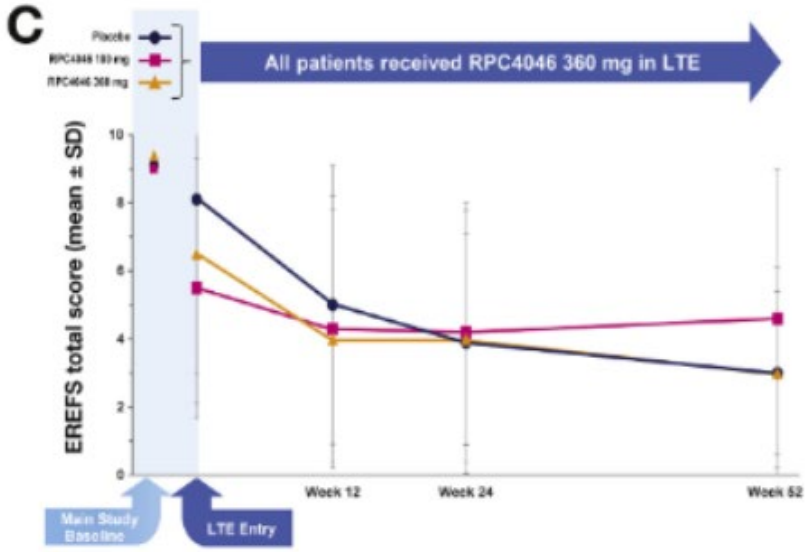
- IL-4 R α antibody (Dupilumab)
  - Reduction of 86.8 eos/hpf vs. placebo, improvement of histological scoring (68.3%), EREFS-score (1.6 points) and dysphagia

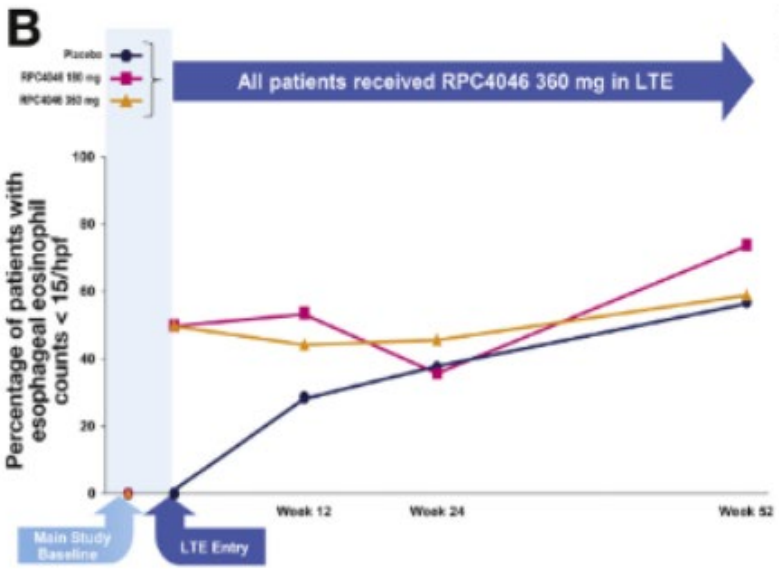
Hirano I et al. Gastroenterology. 2020

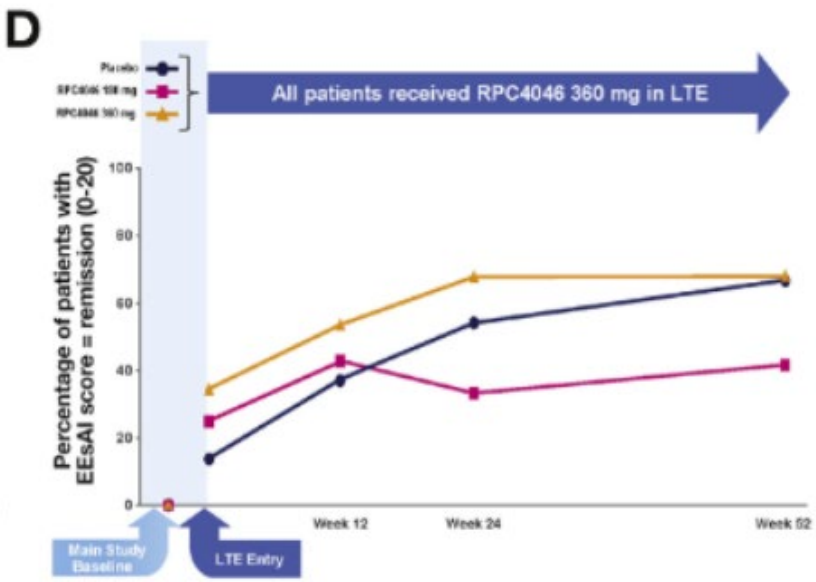
Biologics

- Anti-IL13 anti
  - Significar
    severity, e
  - Numerica
  - Open lab
  - Most com
    infection/i









disease

uction

ogy. 2019
ogy 2021

Biologics

- IL-5Rα (Benralizumab)
  - MESSINA Trial ongoing

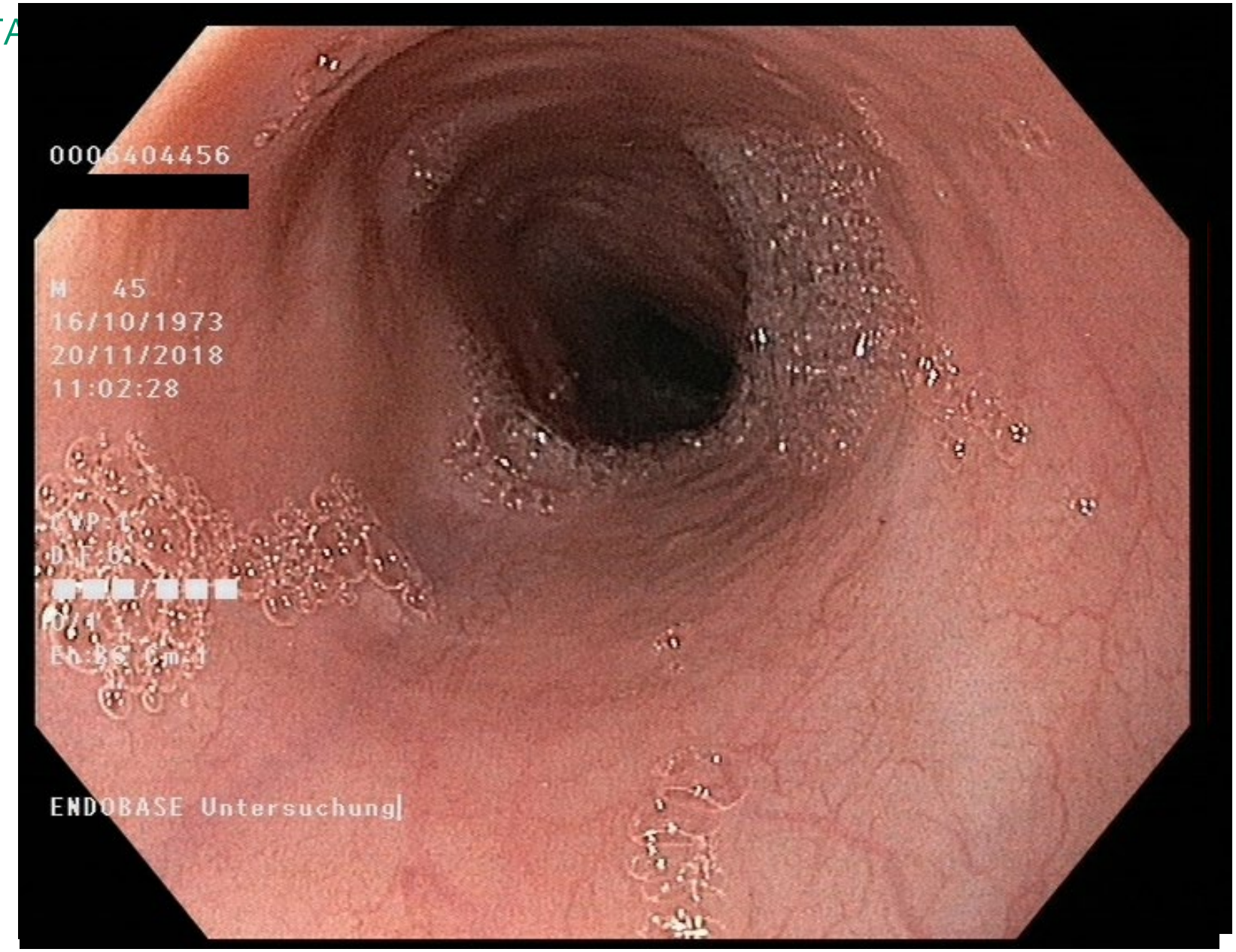
Monitoring

- Symptoms correlate insufficiently with inflammation
- Macroscopic-endoscopic aspect
- EREFS Score 88% Sens. / 92% Spez. Dellon et al. Clin Gastro Hepato, Jan 2016

Endoscopy with biopsy as gold standard

- Data not clear if mucosal healing is necessary (analogous to IBD)

- Control endoscopy 6-12w after treatment changes or otherwise at least 1x/year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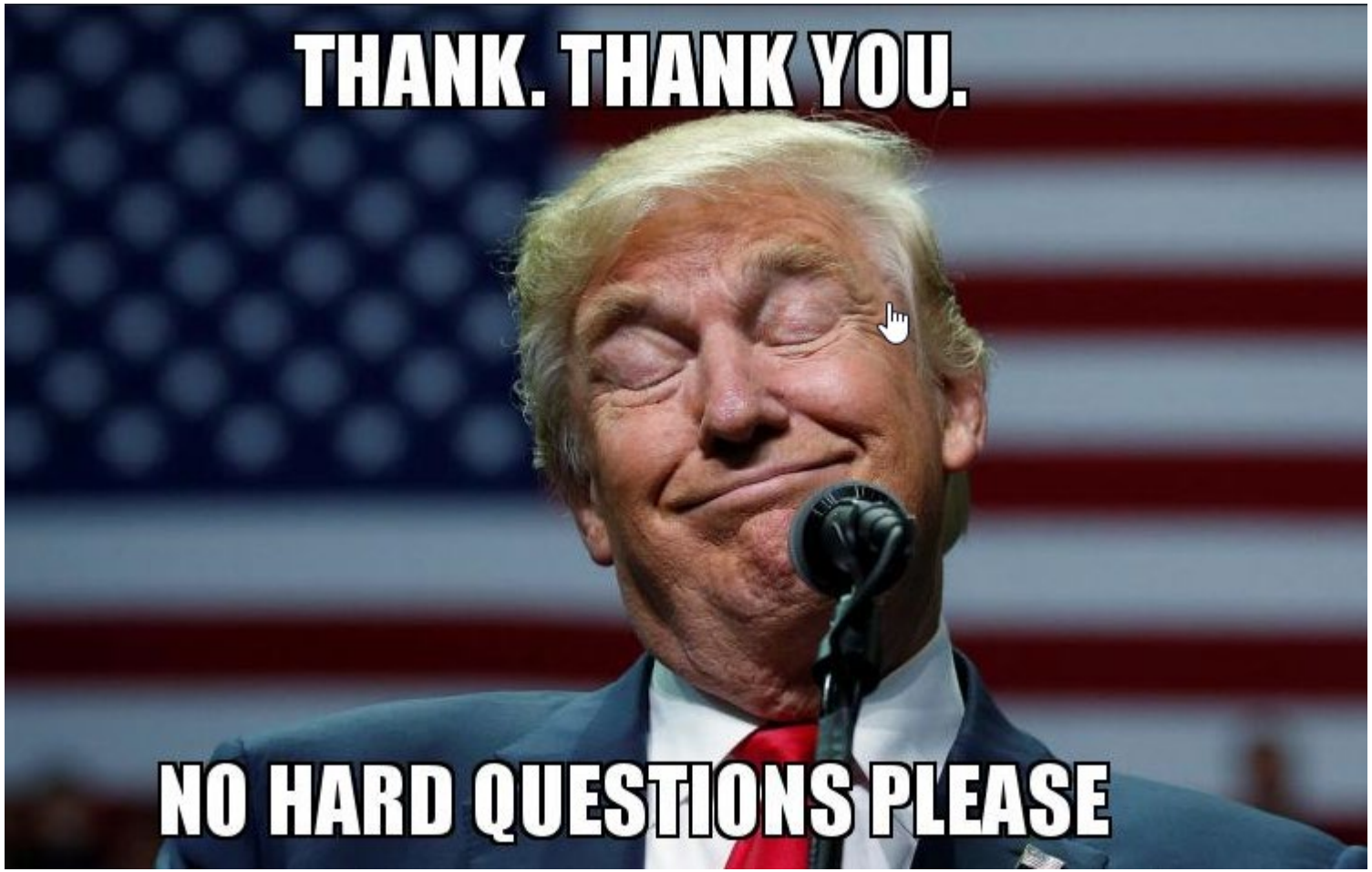
M 45
16/10/1973
20/11/2018
11:02:28
ENDOBASE Untersuchung

Take home messages

- Frequent disease with increasing incidence
- High degree of suffering with relevant potentially life threatening complications
- Simple, effective and safe therapy with good maintenance of remission in most patients
  - PPI, steroids, diet
- Regular control endoscopy for diagnosis and treatment of persistent inflammation or strictures
- Novel, promising therapies


THANK. THANK YOU.
NO HARD QUESTIONS PLEASE

Excursion eosinophilic gastroenteritis

- Epidemiology and pathogenesis similar to EoE

- Characterized through increased tissue eosinophils (esophagus to colon), predilections are stomach and duodenum

Excursion eosinophilic gastroenteritis

- Different clinical signs/symptoms depending on dominant inflammation
  - Mucosal: Abdominal pain, nausea, vomiting, early satiety, diarrhea. Severe cases with malabsorption, protein-loosing enteropathy, malnutrition
  - Muscularis: Dysmotility with obstruction or perforation, nausea, vomiting, distended abdomen, pseudoachalasia
  - Serosal: ascites, additional symptoms as mentioned above

- Therapy
  - Diet
  - Steroids or other immunosuppressants (e.g. AZA, biologics)