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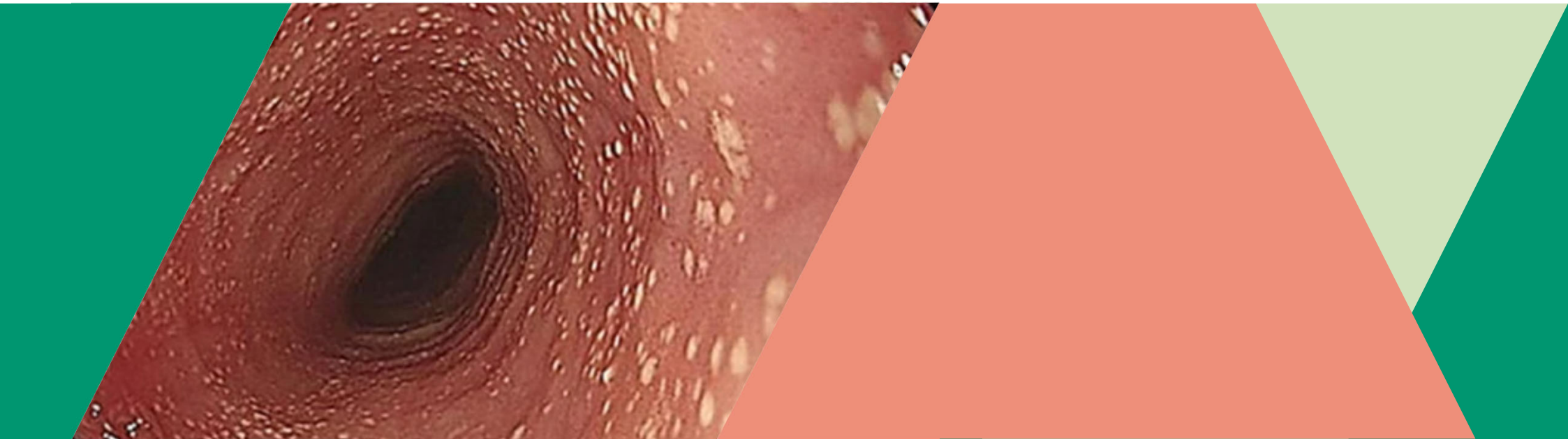
Bible class
Eosinophilic esophagitis (EoE)

01.03.2022, M. Knecht



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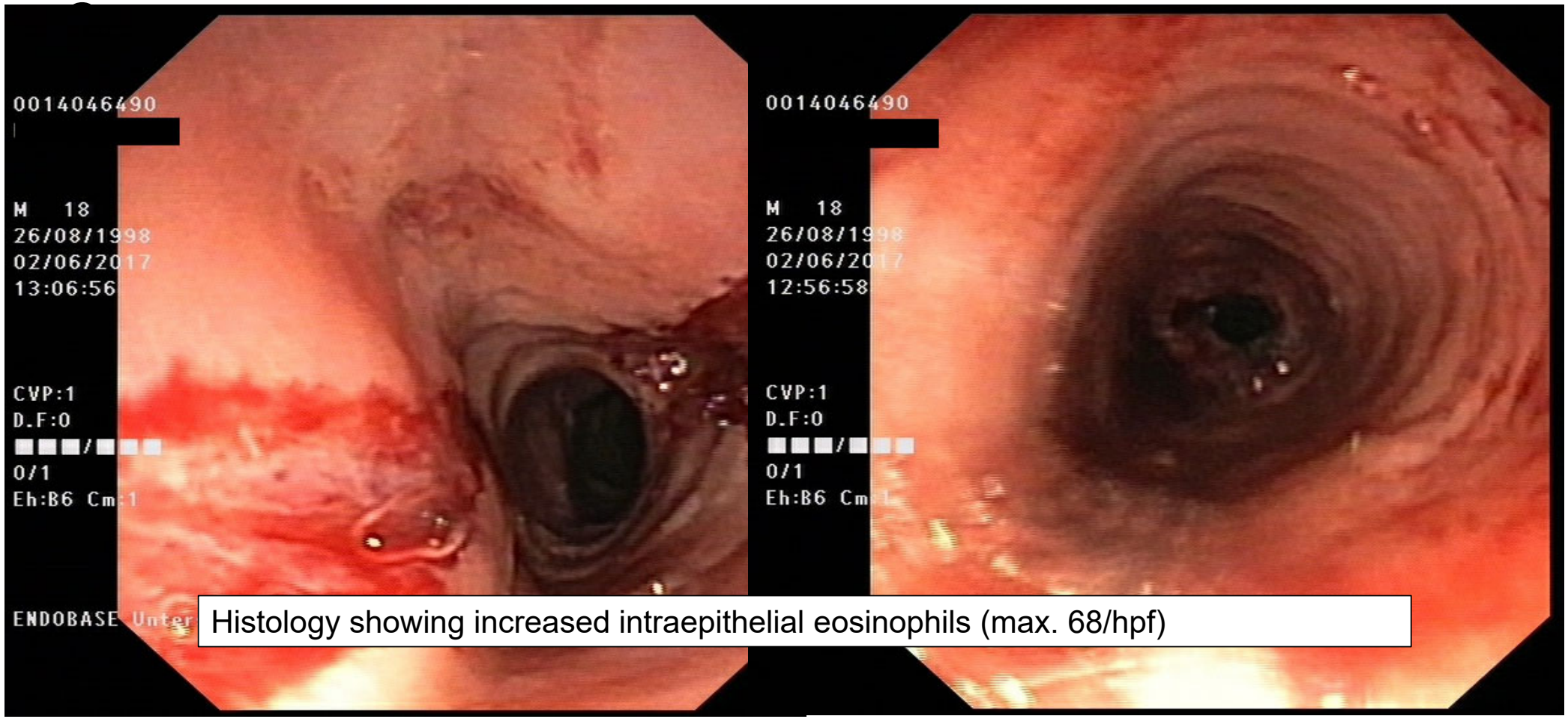
Case

- 18yo patient

- Recurring food impaction and chest pain while eating
- Always been a slow eater

- Personal history
  - Asthma bronchiale
  - Allergy to soy and eggs



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

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Case

- Further workup – apart from ineffective motility - negative (pH-metry, manometry, barium swallow)
- Diagnosis of eosinophilic esophagitis
- Treatment with high dose PPI

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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)

T-cell mediated food allergy

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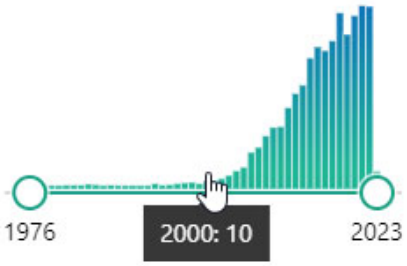
History of eosinophilic esophagitis (EoE)

- First description of a case in 1970, misinterpreted as motility disorder
- Recognition as a disease in the early 90ies
  - Alex Straumann et al, Switzerland (Spital Olten). Published 1994 in SMW
  - Stephen Attwood et al, USA. Published 1993 in Digestive Disease Science
- Increasing interest after 2000
- First guidelines in 2007



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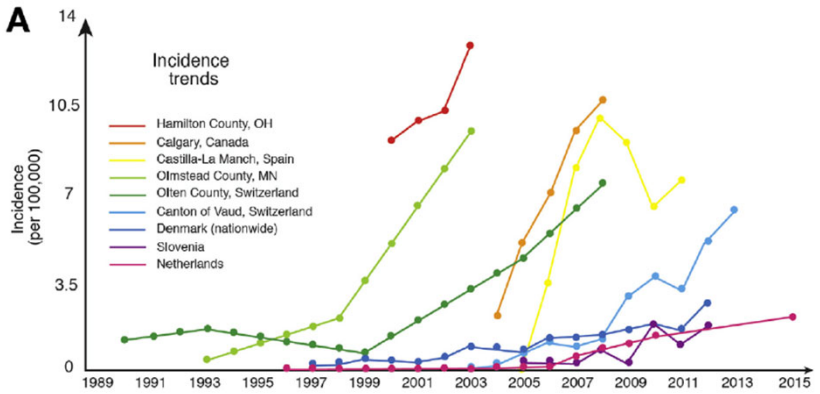
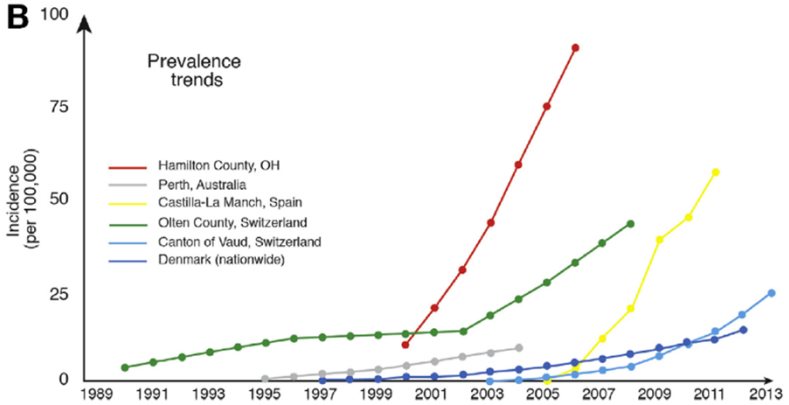
Epidemiology



- Prevalence
  - 32.5 adult, resp. 30.9 pediatric patients per 100’000 persons



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

Giriens B et al. J Allergy Clin Immunol 2011
Arias A et al. APT 2016
Molina-Infante J et al. United European Gastroenterol J. 2018
Dellon E, Hirano I et al. Gastroenterology 2018

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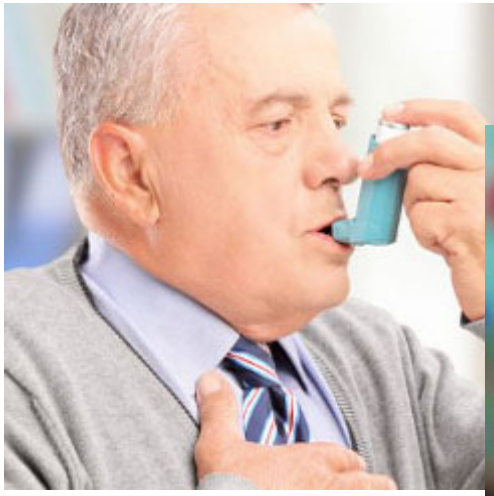
Classical patient

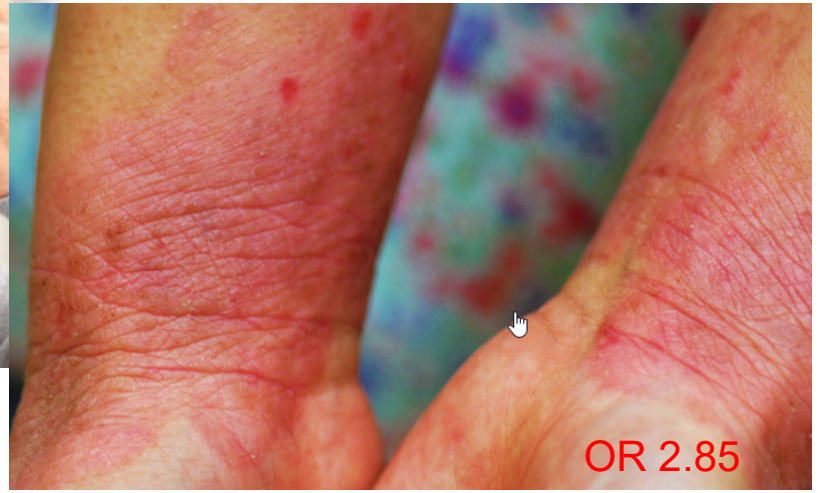
- Predominantely men (ratio 3:1)
- Median age around 30y, rarely old age
- Caucasian
- Urban-rural, north-south/west-east gradient
- History of atopy

Risk factors

- **Atopy**

50-60% EoE patients are atopic and sensitized to food and aeroallergens
Surge in pollen season





González-Cervera J et al. Ann Allergy Asthma Immunol, 2017 May

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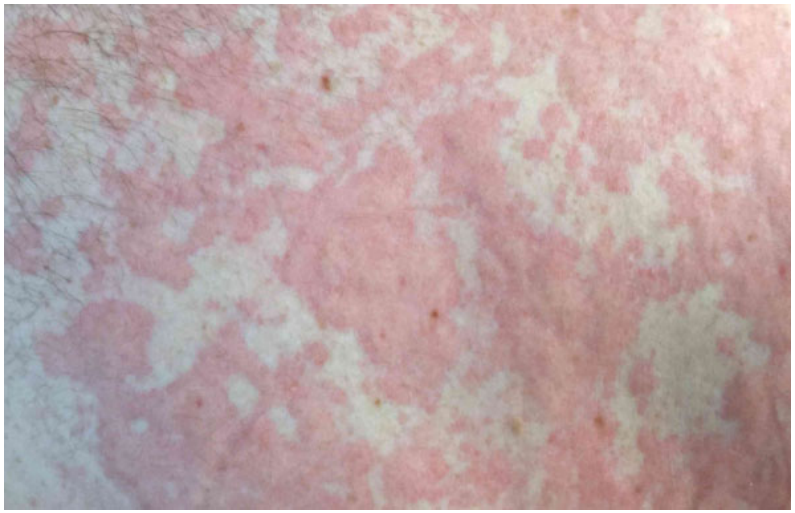
Risk factors



- 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/pollen
  - Risk of 2.72% for de novo EoE

Miehlke S et al. Case Rep Gastroenterol 2013
Lucendo AJ. Ann Allergy Asthma Immunol 2014

Potentially predictive factor of subsequent EoE

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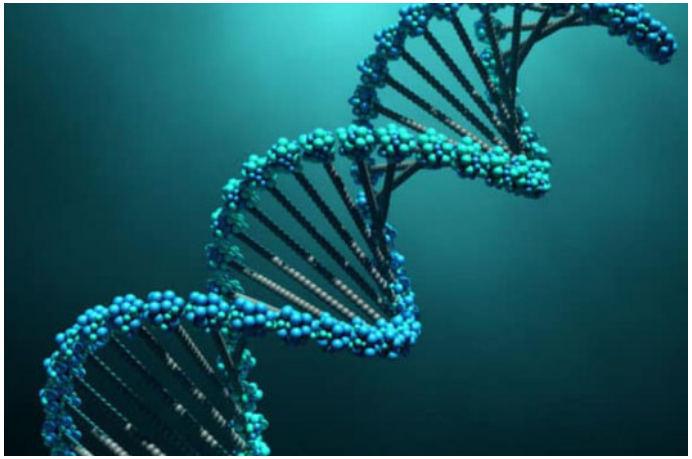
Risk factors



- **Genetics and familial clustering**
  - Thymic stromal lymphopoetin (TSLP) -> Th2 differentiation
  - CCL26 -> Production of eotaxin-3
  - CAPN14 -> IL-13 induced cystein proteinase, esophagus specific
  - Shared genetics with UC, PSS and MS

Often in atopics

EoE-specific

- Genetic inheritance
  - Relative risk 10 to 64x increased (higher in male relatives, esp. brothers)
  - 1.8 – 2.4% in relatives

Collins MH. Gastroenterol Clin North Am. 2014
Alexander ES et al. J Allergy Clin Immunol 2014
Peterson K et al. Am J Gastroenterol. 2016

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.

Neither necessary nor sufficient for EoE, but modulate risk

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.

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Further risk factors

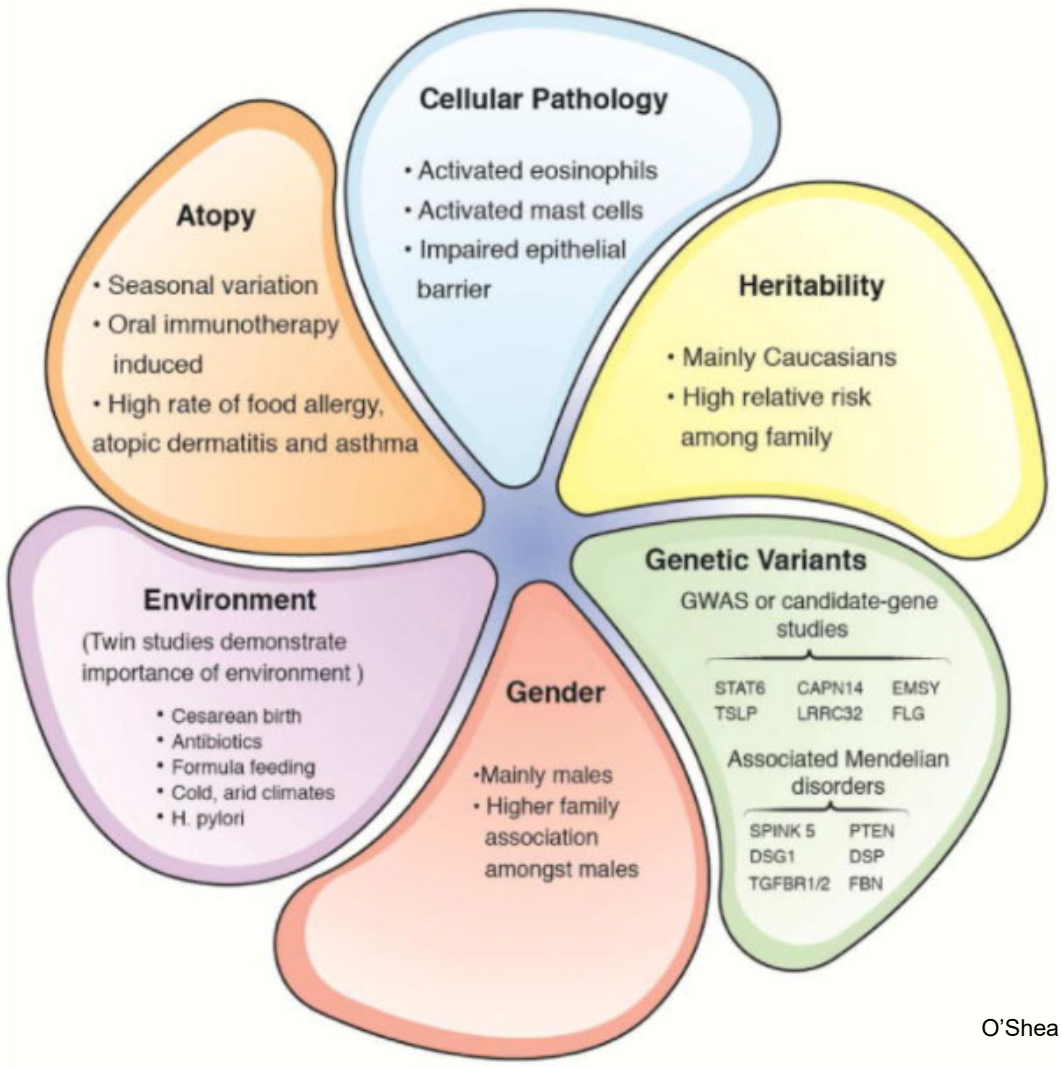
- **Developmental/environmental**
  - C-section
  - Premature birth
  - Antibiotics and PPI in childhood
  - No breastfeeding/formula
  - Cold and dry climate
  - H.pylori (Inversely proportional!)

Modulation of microbiota

↑ Firmicutes and Proteobacteriae
↓ Streptococci

Benitez AJ et al. Microbiome 2015

Skewing toward Th2-inflammation type
Similar to atopic diseases



O'Shea KM. Gastroenterology. 2018 Jan

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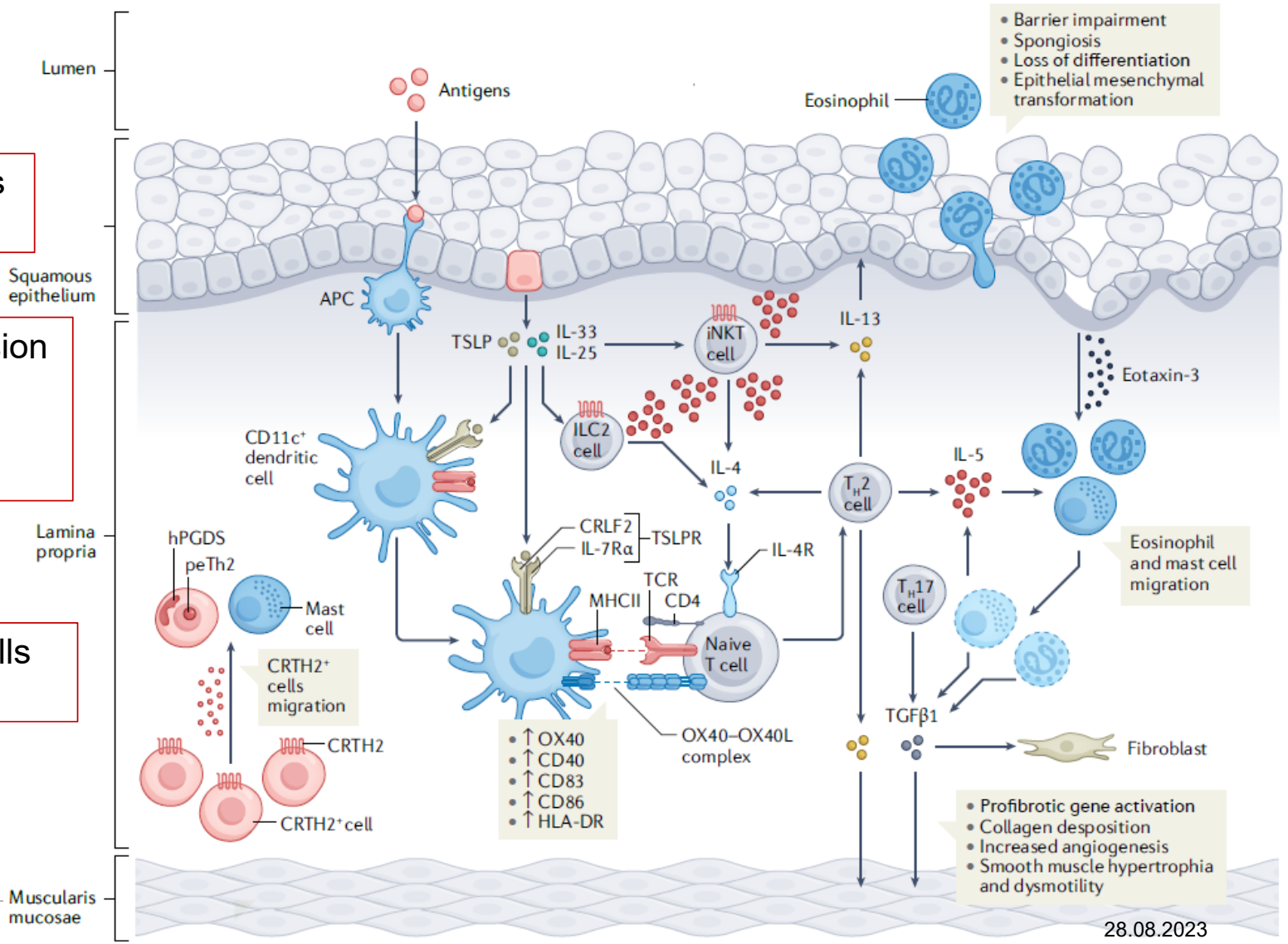
Penetration of antigens as primary driving factor

- Primary impaired expression of barrier proteins
- IL-13 driven epithelial dysfunction

- Increase in IgG4-pos. Cells and systemic IgG4



Clayton F et al. Gastroenterology 2014 Sep
Weidlich S et al. J Clin Gastroenterolo 2020 Jan
Kosaka S et al. Dig Dis Sci 2021

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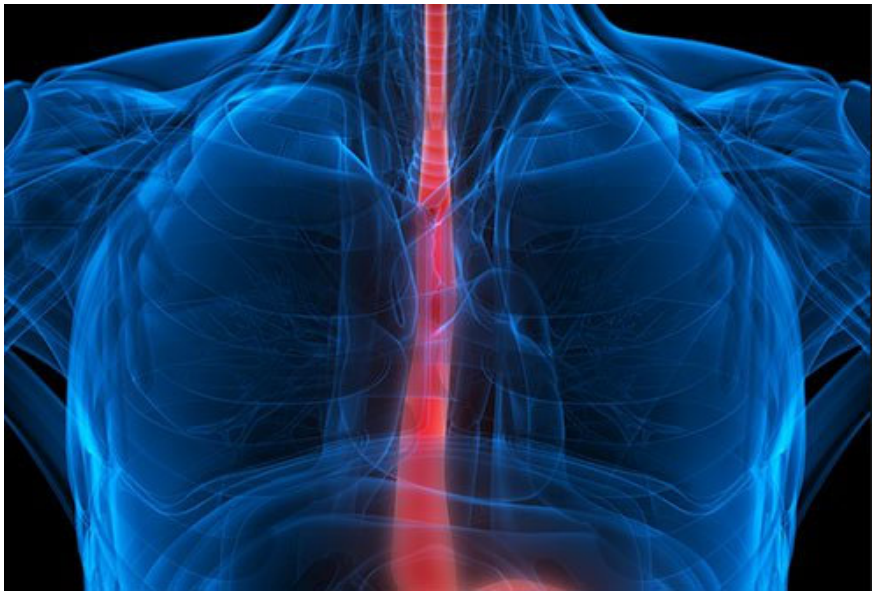
Symptoms



- Adults
  - Dysphagia (~75%)
  - Impactions (33-54%)
  - Retrosternal pain or burning (>50%)
  - 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

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Symptoms

- Children
  - Reflux/regurgitation
  - Refusal or avoidance of food
  - Reduced growth
  - Nausea and vomiting
  - Abdominal pain
  - Diarrhea

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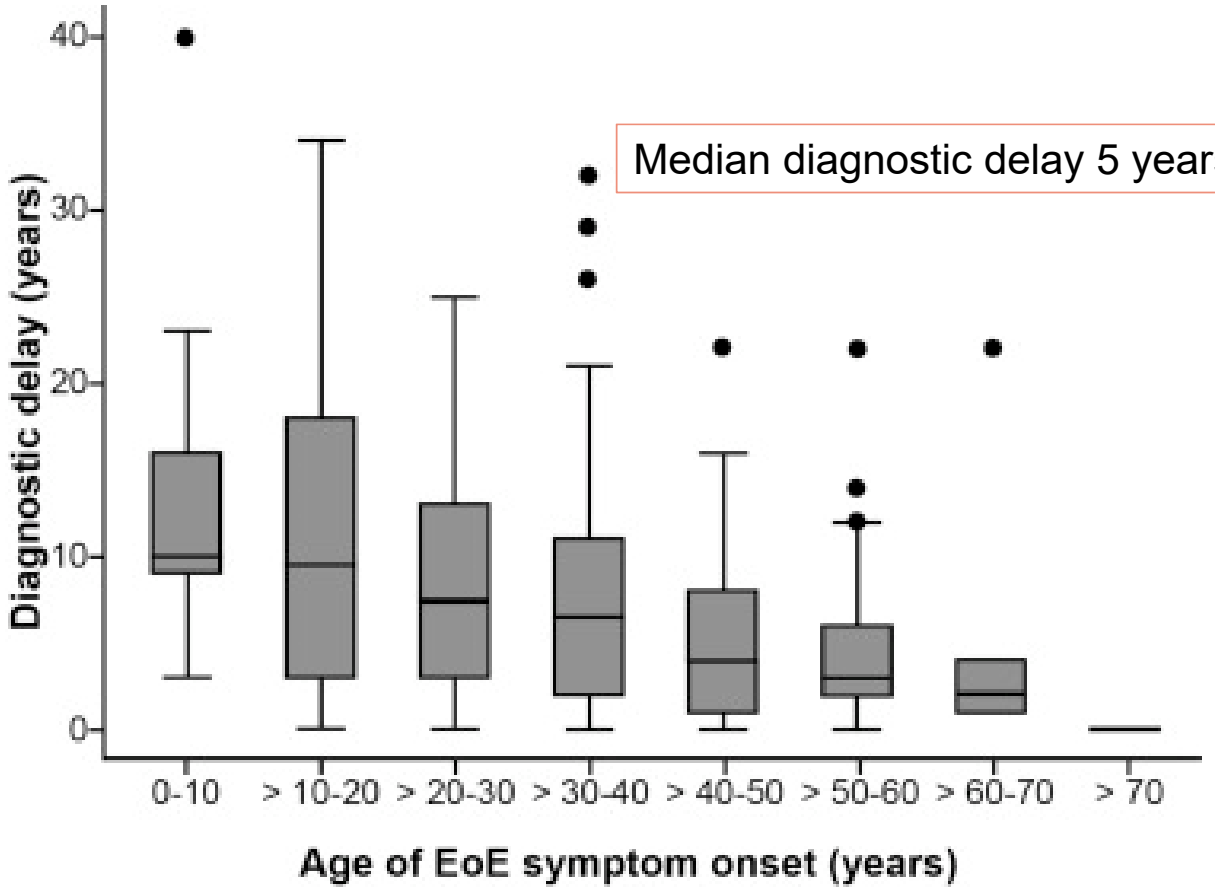
Symptoms

- Prevalence of EoE
  - Esophageal sy
  - Dysphagia
  - Impactions



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

Schoepfer AM et al, Gastroenterology 2013;145:1230

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Diagnosis

- Combination of symptoms, endoscopy, histology

- Endoscopy with biopsies
  - 6-8 biopsies, different locations, most importantly mucosal changes
  - Oral and aboral (seperate)
  - Even when normal mucosa (<5% of patients)

Hirano I et al. Gut 2013

  - PPI withdrawal >3w prior

- Biopsies stomach/duodenum to exclude EGID

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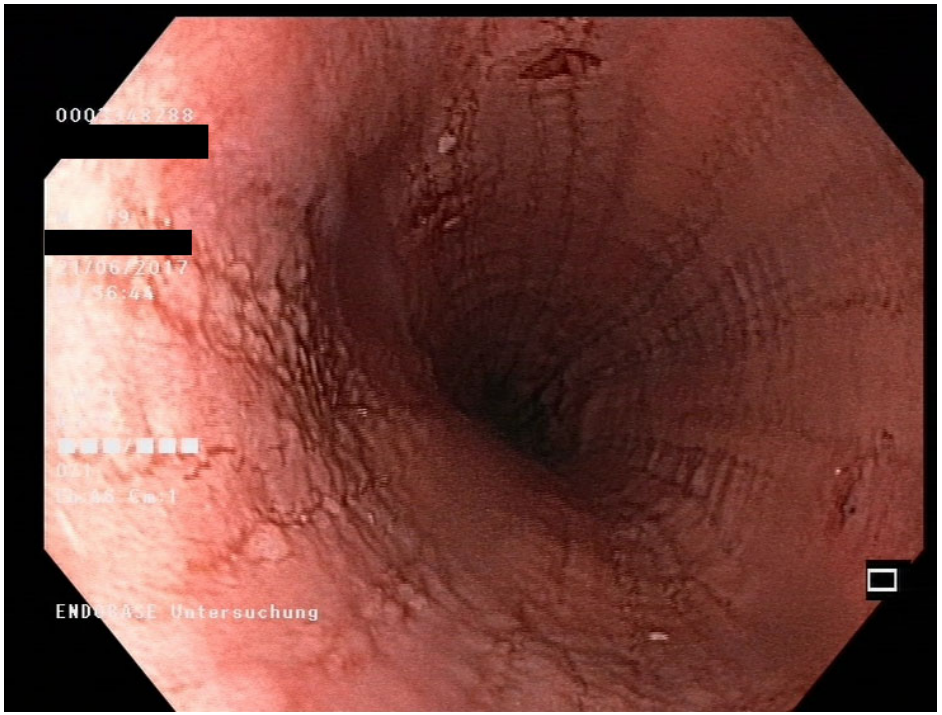
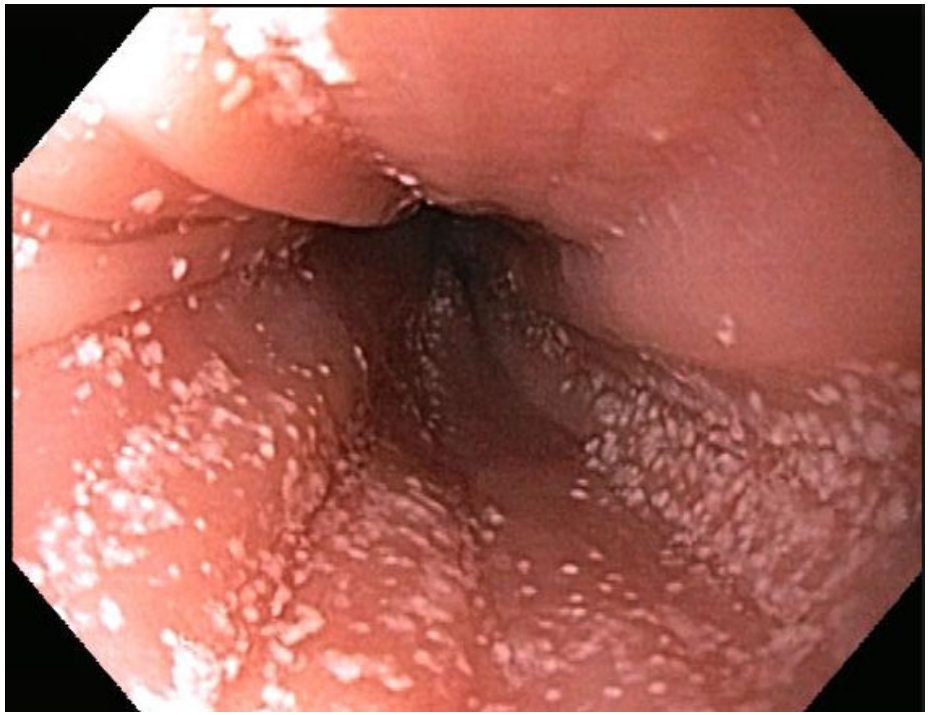
Endoscopy





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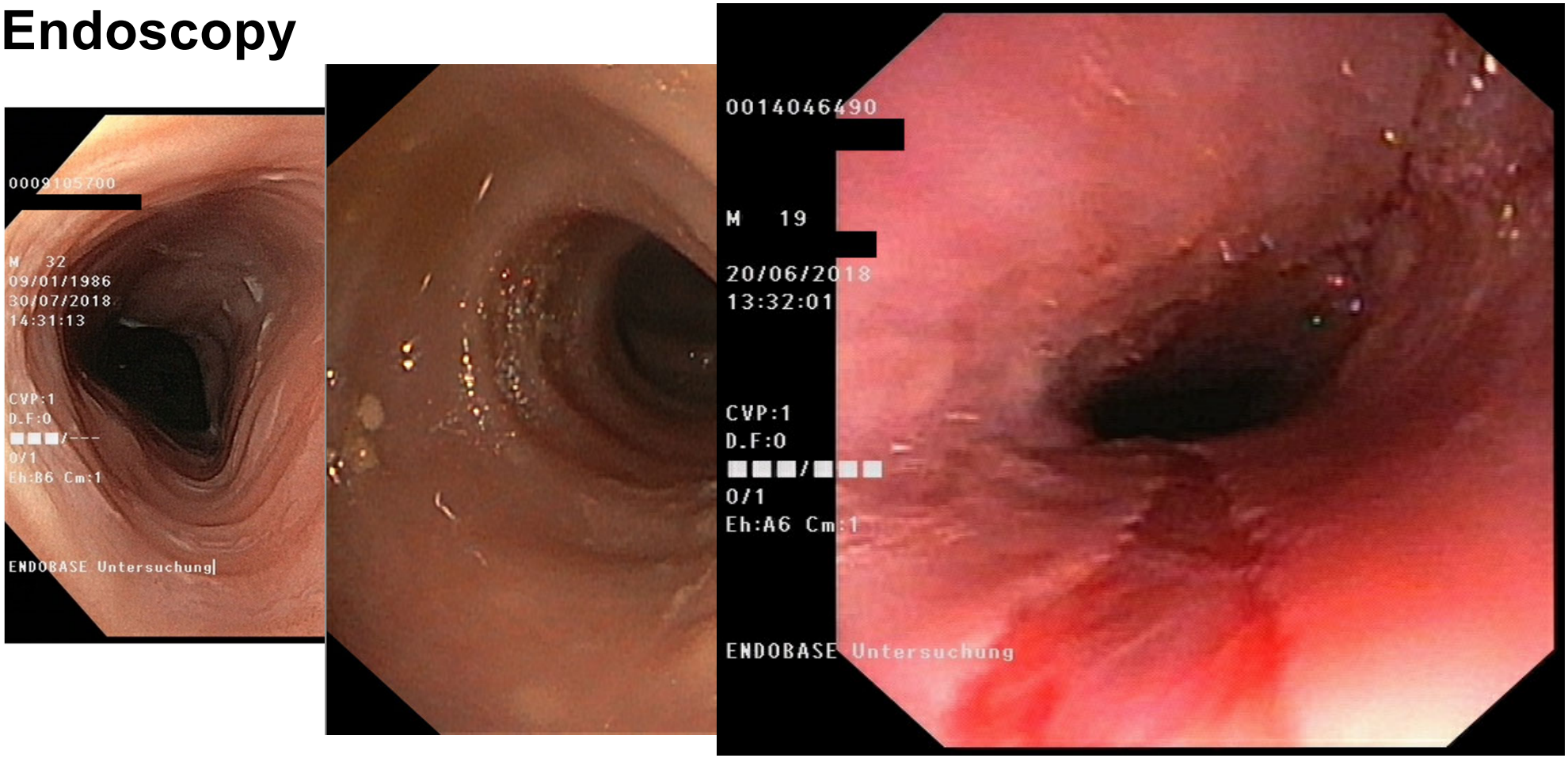
Endoscopy



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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

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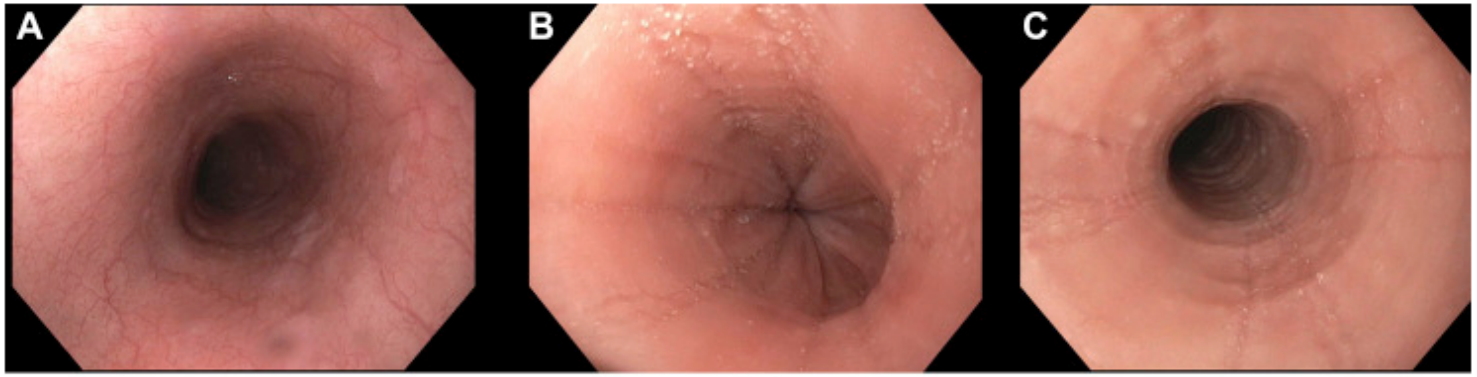
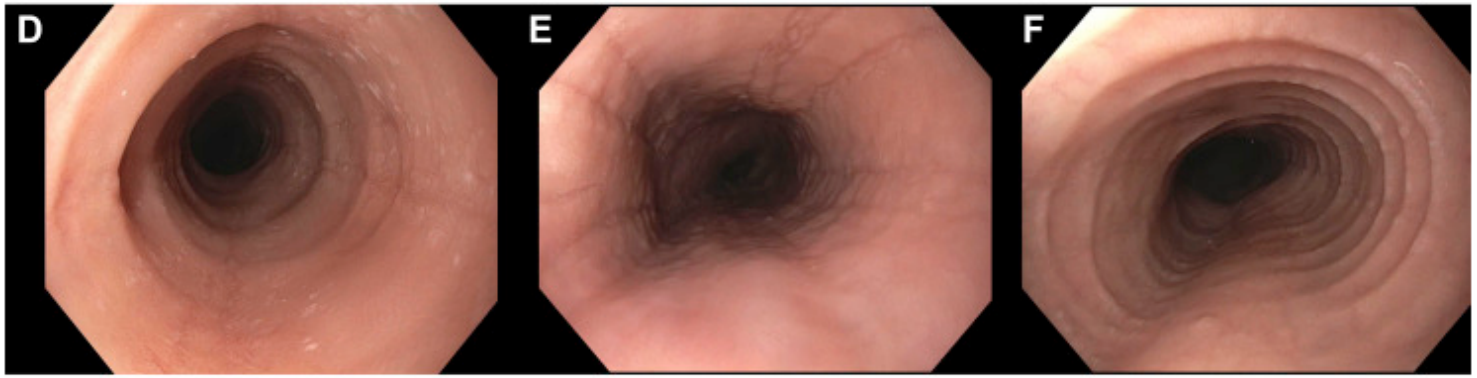
EREFS





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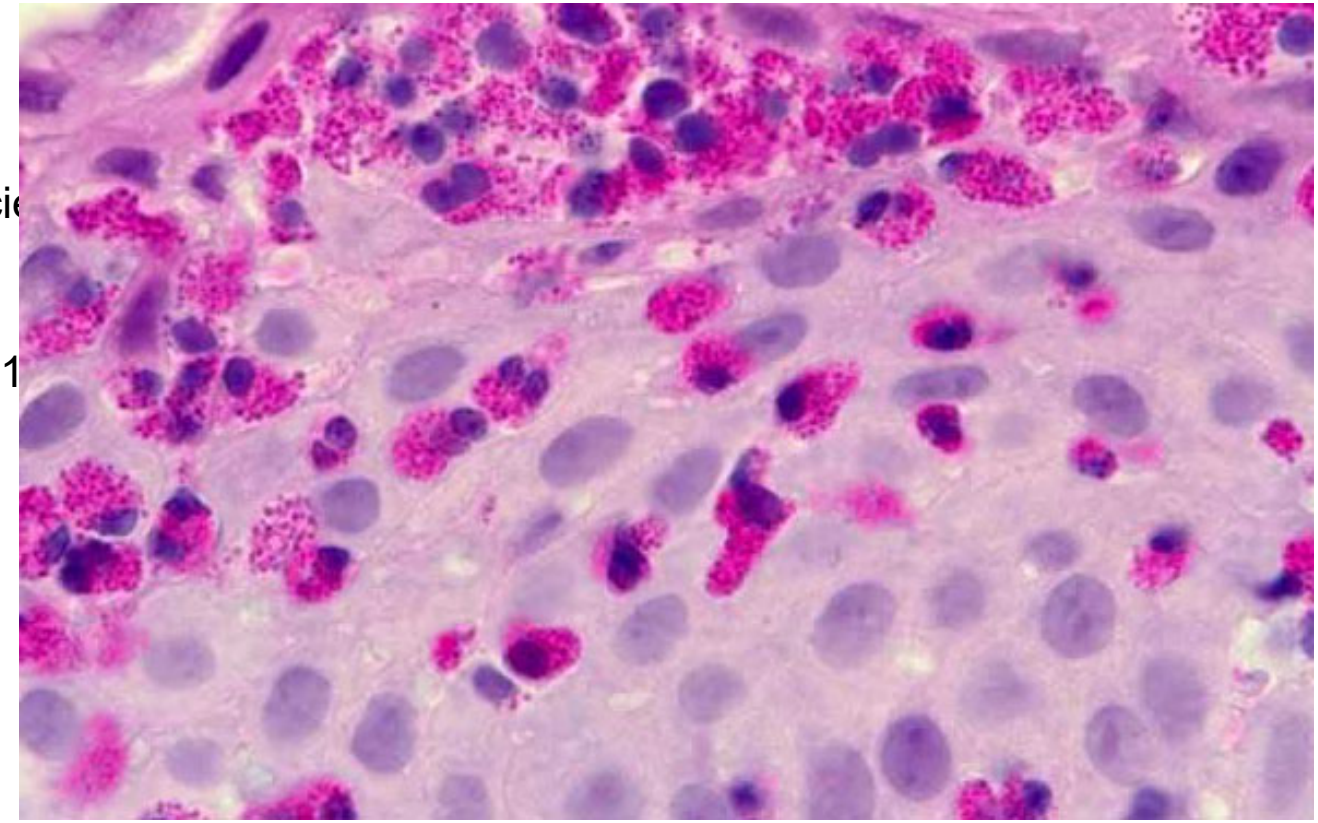
Histology

- H&E stain sufficie
- Histology with ≥1



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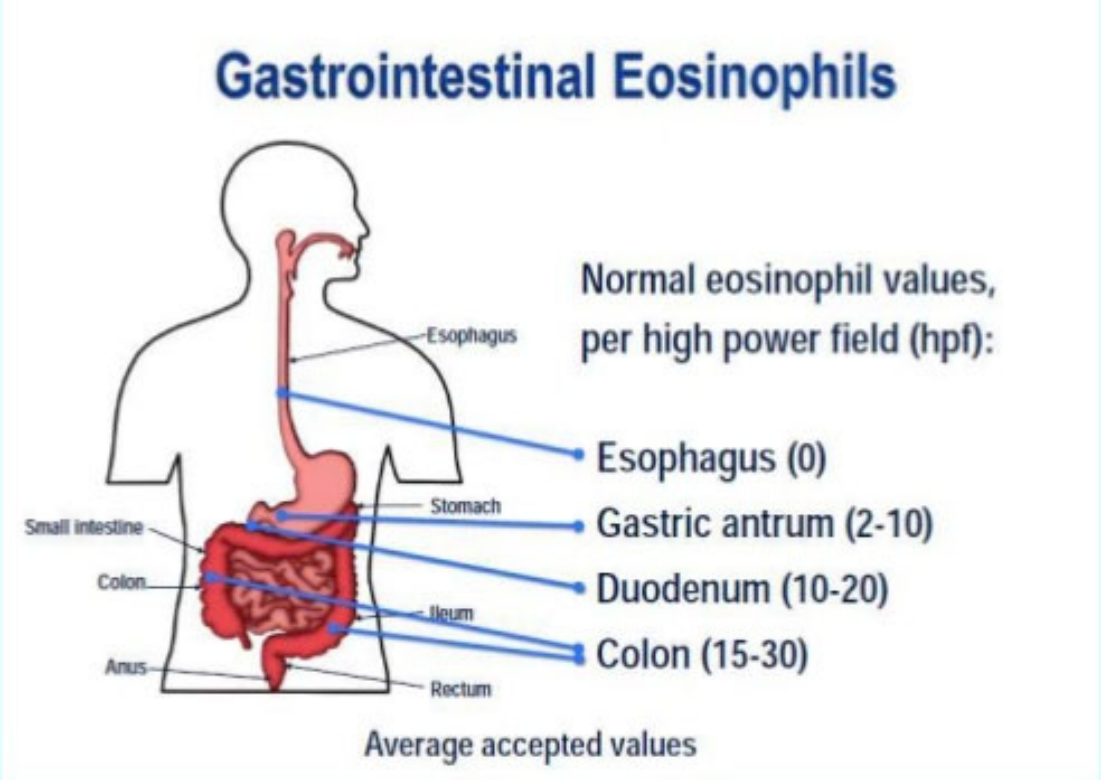
Normal distribution of eosinophils in GIT



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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

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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

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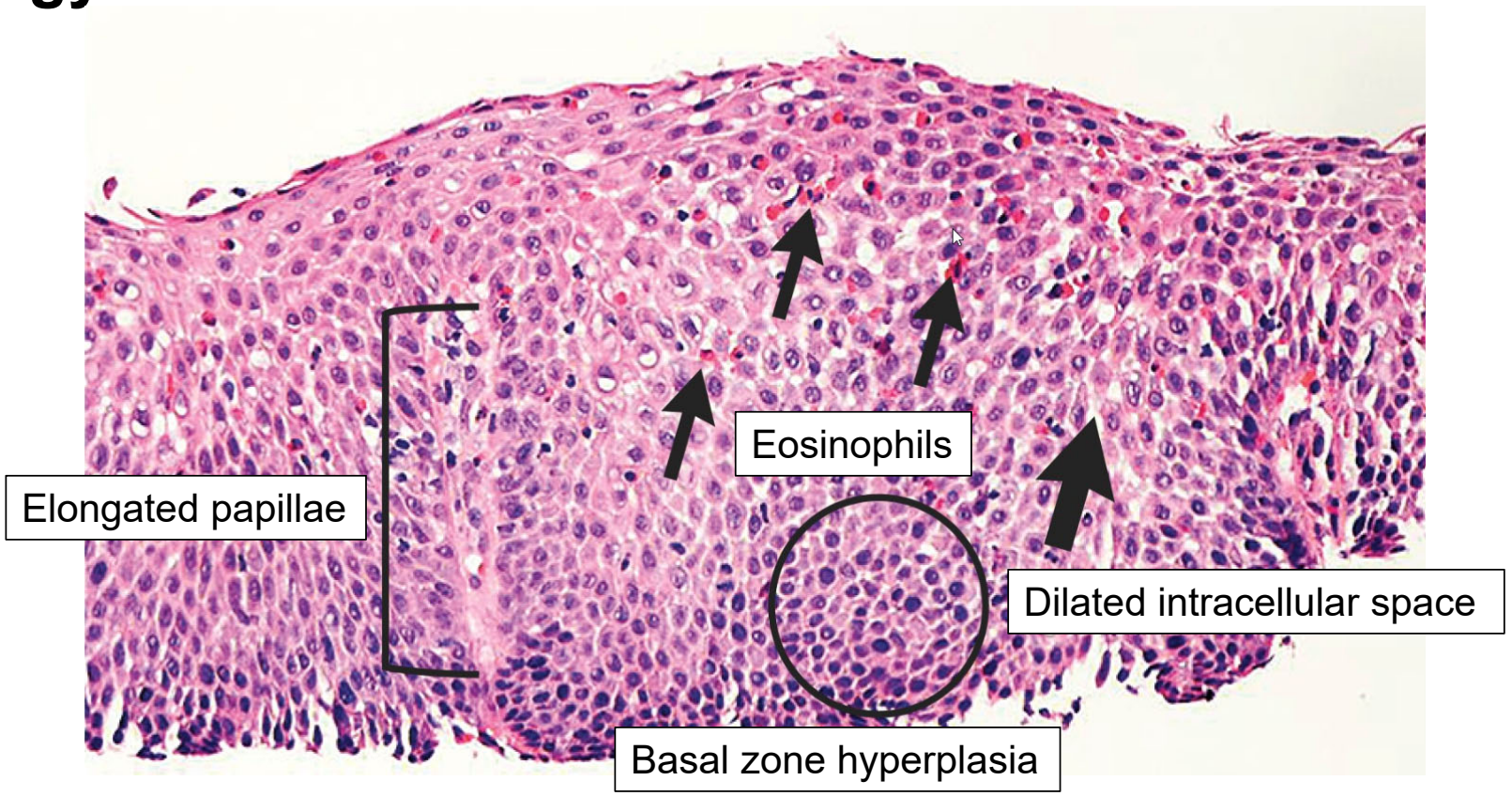
Histology



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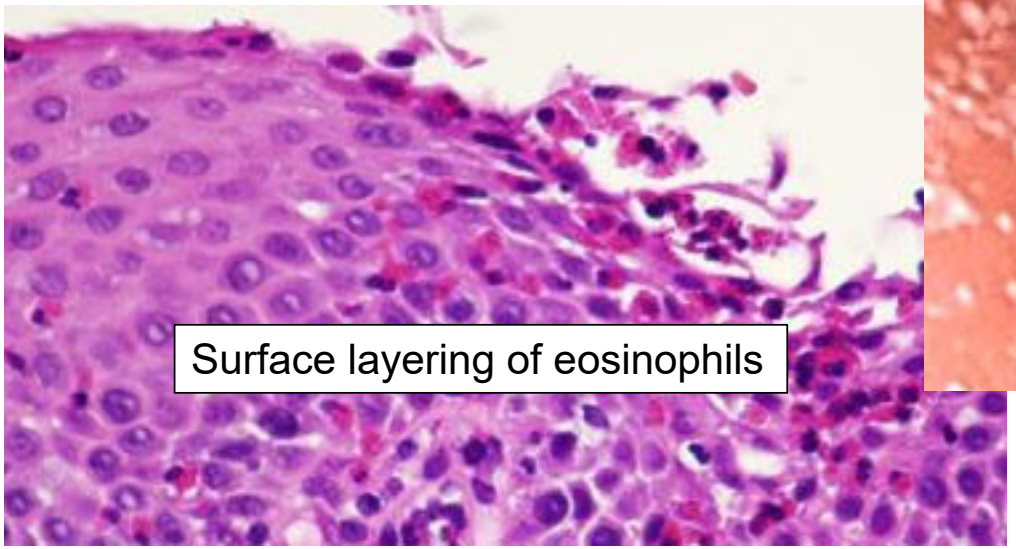
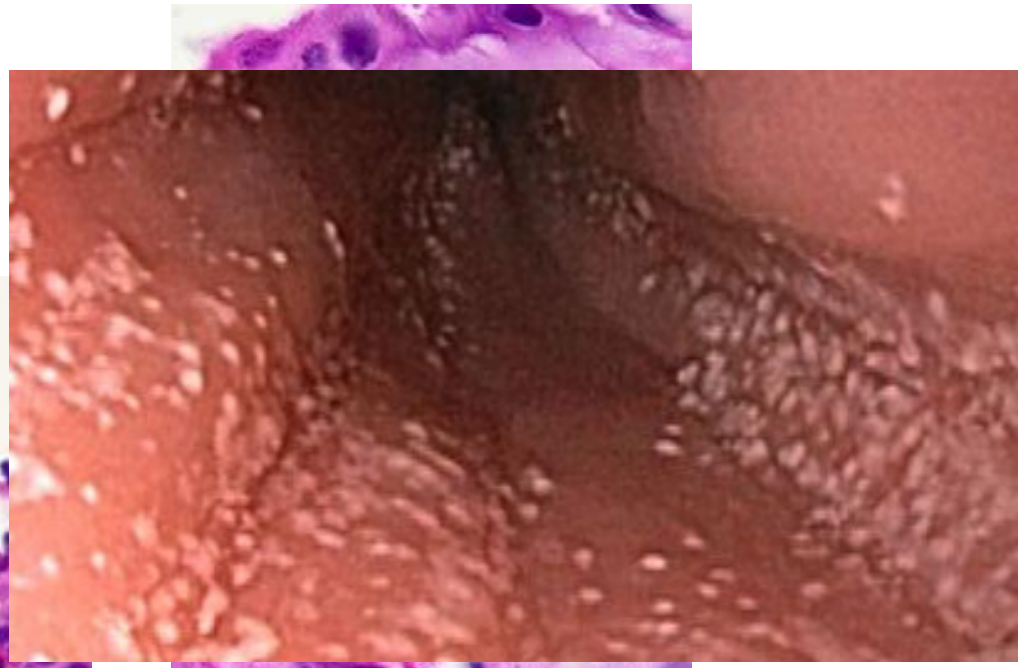
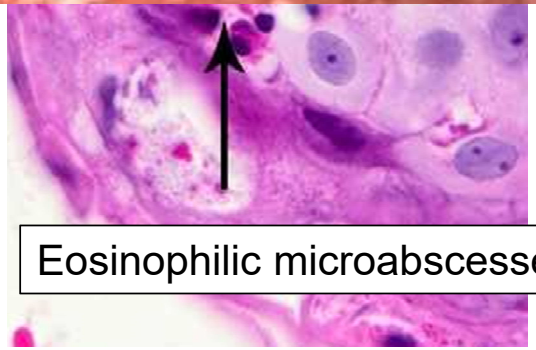
Histology





EoE>GERD



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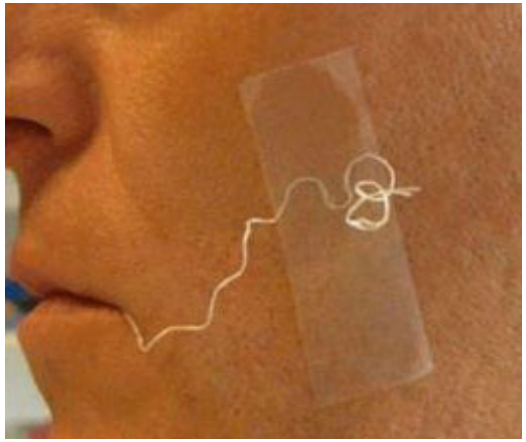
Non-invasive markers

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

Katzka D et al. Am J Gastroenterol. 2017

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

Ackermann S et al. Am J Gastroenterol. 2019



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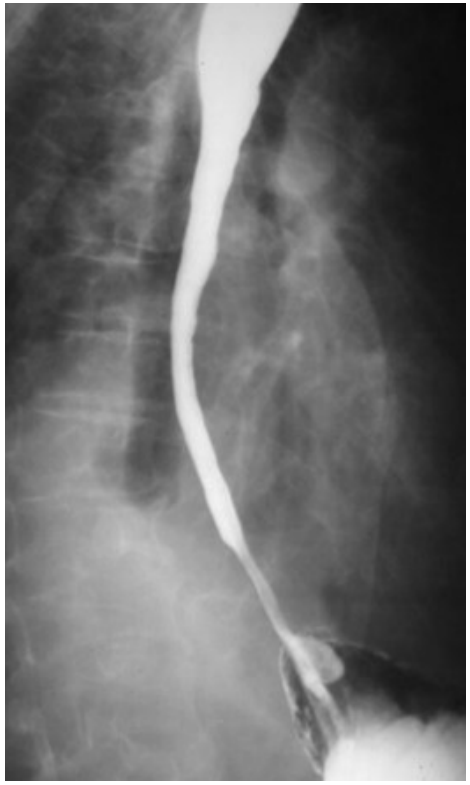
Radiology/barium swallow

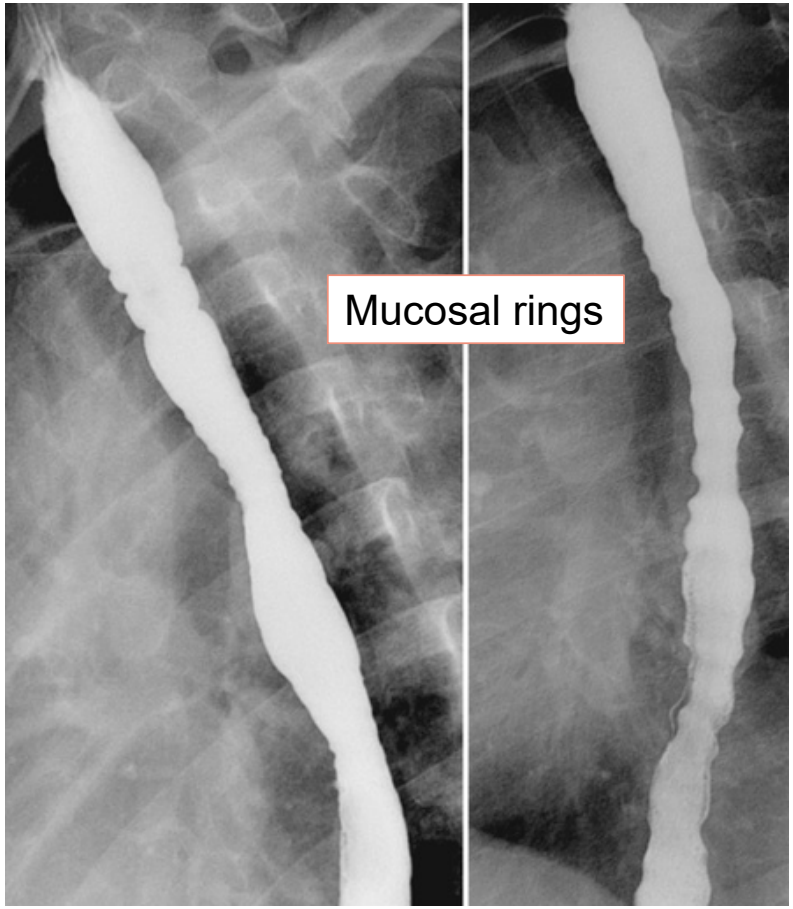
- Not helpful in diagnostics

- BUT

  -> Important for stenoses

  - Amount
  - Location
  - Extension and diameter





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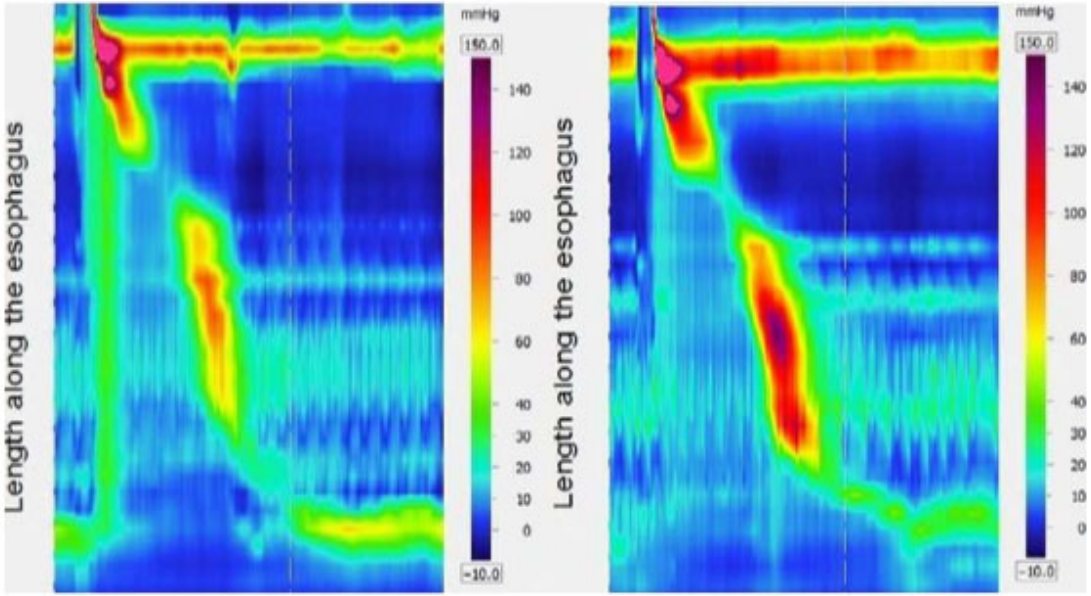
HR-manometry

- 1/3 with altered motility
- 86% improve after therapy

- Frequent findings
  - Early, panesophageal increase in pressure
  - Weak and insufficient peristalsis



- Possibly when persistent symptoms despite remission and absence of fibrosis

Nennstiel S et al.. Neurogastroenterol Motil 2016
Roman S et al. Neurogastroenterol Motil. 2011 Mar

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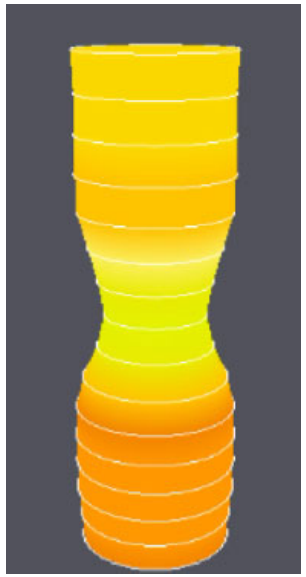
FLIP

- Accurate assessment of esophageal distensibility
- Monitoring of
  - ➢ progression of fibrosis
  - ➢ Response to therapy

Kwiatek M et al. Gastroenterology 2011



Impedance

- Lower resistance in active EoE
- Significant inverse correlations between MI and eos/HPF
- Cut off 2300 Ω for active EoE
  - ➢ 90% sens and 91% spec

Katzka DA et al. Clin Gastroenterol Hepatol 2015

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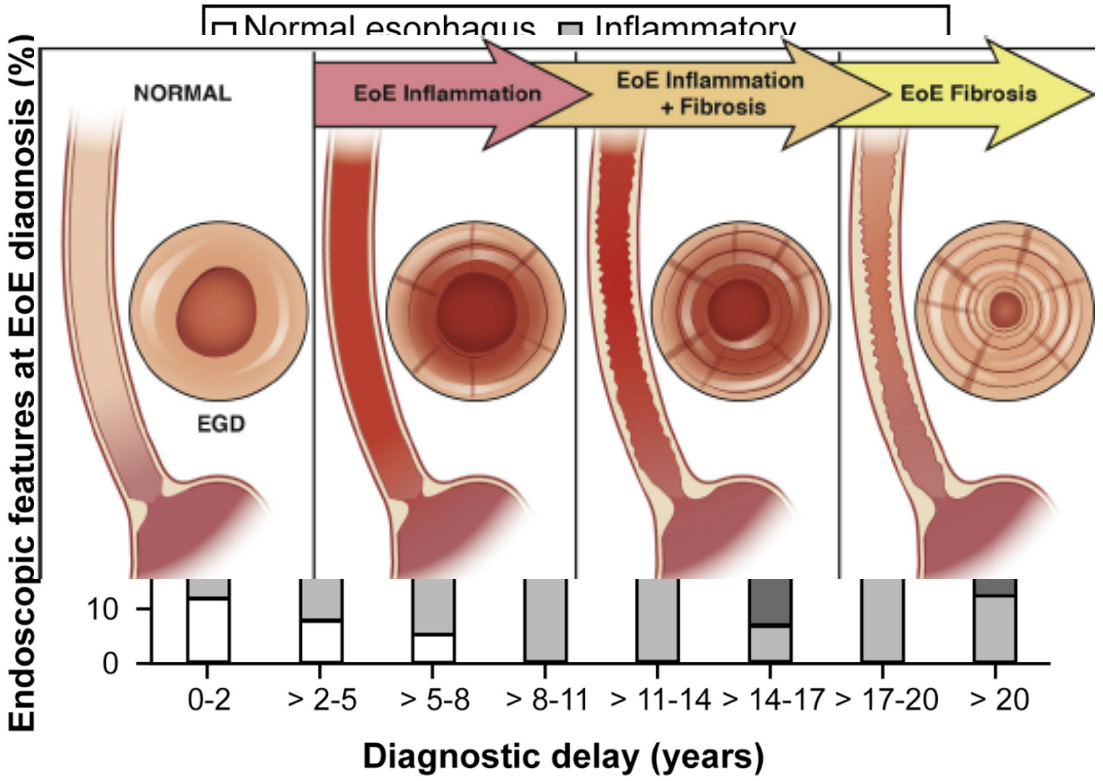
Natural course

- Chronic progressive inflammation
- Fibrotic tissue remodelling
- Strictures



Schoepfer AM et al, Gastroenterology 2013;145:1230

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Complications

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

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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 et al. J Pediatr Gastroenterol Nutr. 2018 Aug
Molina-Infante J et al. 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. Clin Gastroenterol Hepatol 2016;14:13–22

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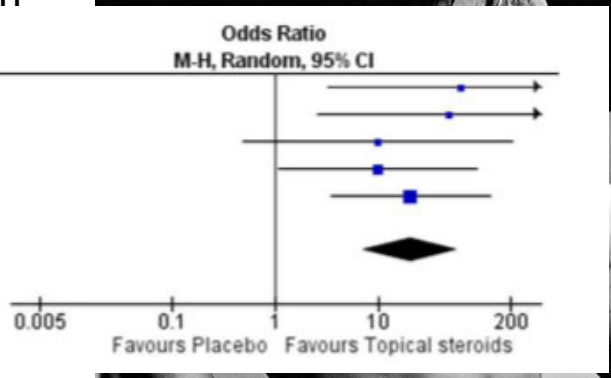
Steroids

- Highly effective in induction and maintenance of remission
  - 66%,
- Topic Buc

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

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Steroids

Table 6. Proportion of Patients With Persistence/Recurrence of EE Symptoms by Visit Week

	Week 4	Week 12	Week 18	Week 24
Prednisone	0.0% (0/32)	11.1% (3/27)	35.7% (10/28)	44.4% (12/27)
Fluticasone	2.8% (1/36)	3.6% (1/28)	21.7% (5/23)	45.8% (11/24)
Chi-square *P* value	.3422	.2817	.2758	.9207

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Steroids

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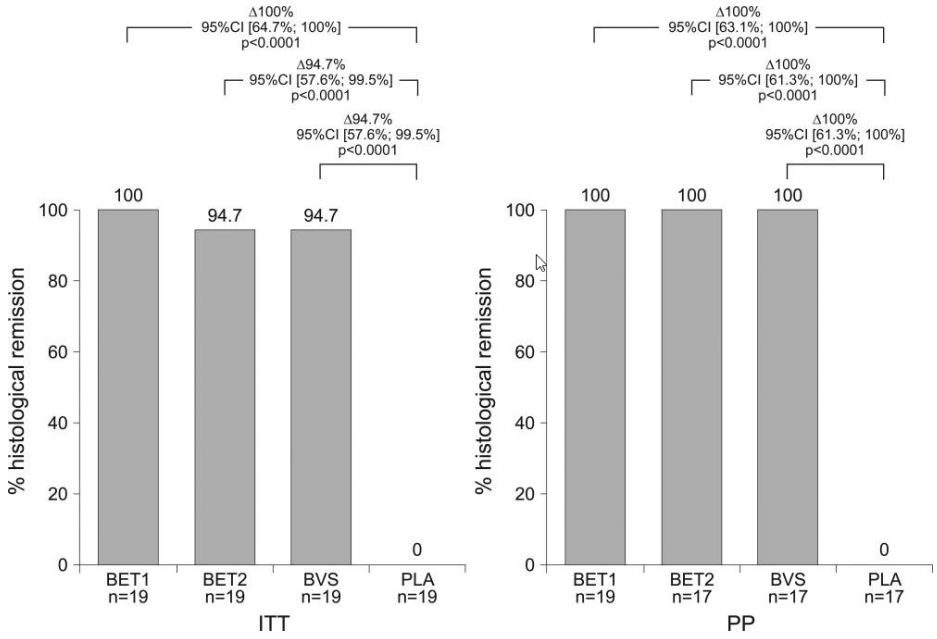
	NEB (n = 11)	OVB (n = 11)	p value
Primary outcomes			
Overall eosinophil counts (eos/hpf ± SD)			
Baseline maximum eosinophil count	101 ± 85	83 ± 89	0.62
Baseline mean eosinophil count	23 ± 20	20 ± 24	0.80
Post-treatment max eosinophil count*	89 ± 94	11 ± 23	0.02
Post-treatment mean eosinophil count*	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

Dellon ES et al. Gastroenterology 2012

Steroids

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



Mielke S et al. Gut. 2016 Mar

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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) with continuing steroids

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

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Nutrition

- Elemental diet
  - Only after therapy beforehand
  - Response rate 90% (children and adults)
  - Clinical improvement in 8.5±3.8 d
  - Histological remission i
- BUT
  - Taste take to (cooling, straw), sufficient compliance only in 1/3
  - QOL, social ilation
  - Costs (also endoscopies)

Efficient, but rather impractical in the long term

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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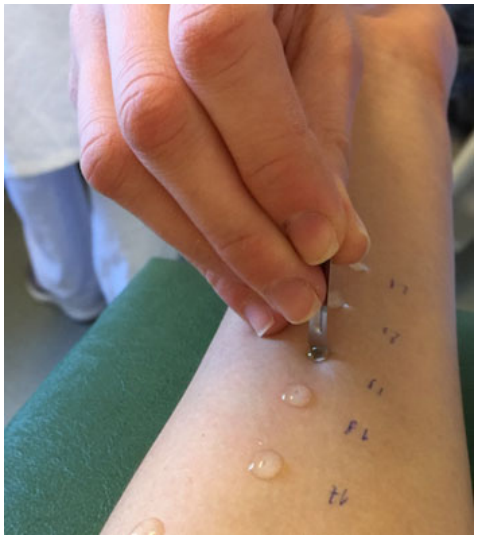
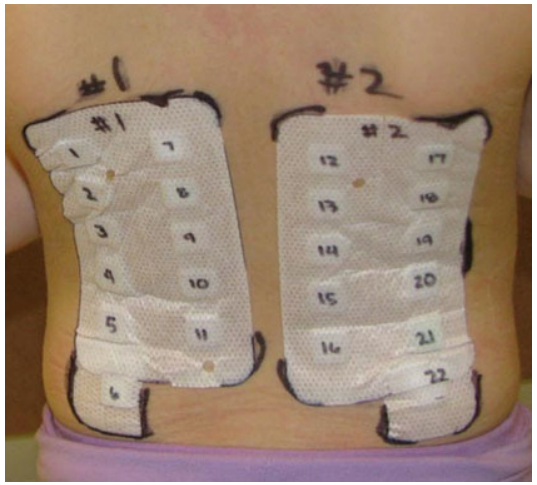
Allergy testing

- 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%)





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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 et al. J Allergy Clin Immunol. 2002 Feb

- 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 et al. Allergy. 2014

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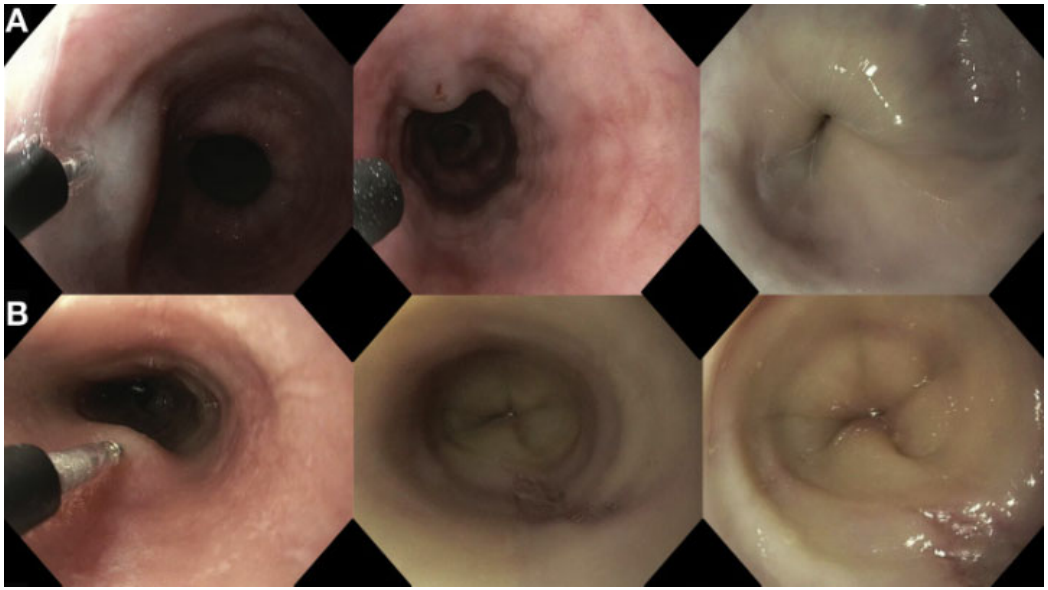
Future testing?

- Direct esophageal prick test



Warners MJ et al. Gastroenterology 2018

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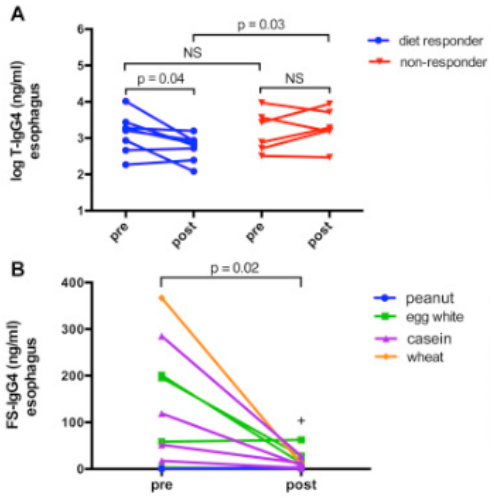
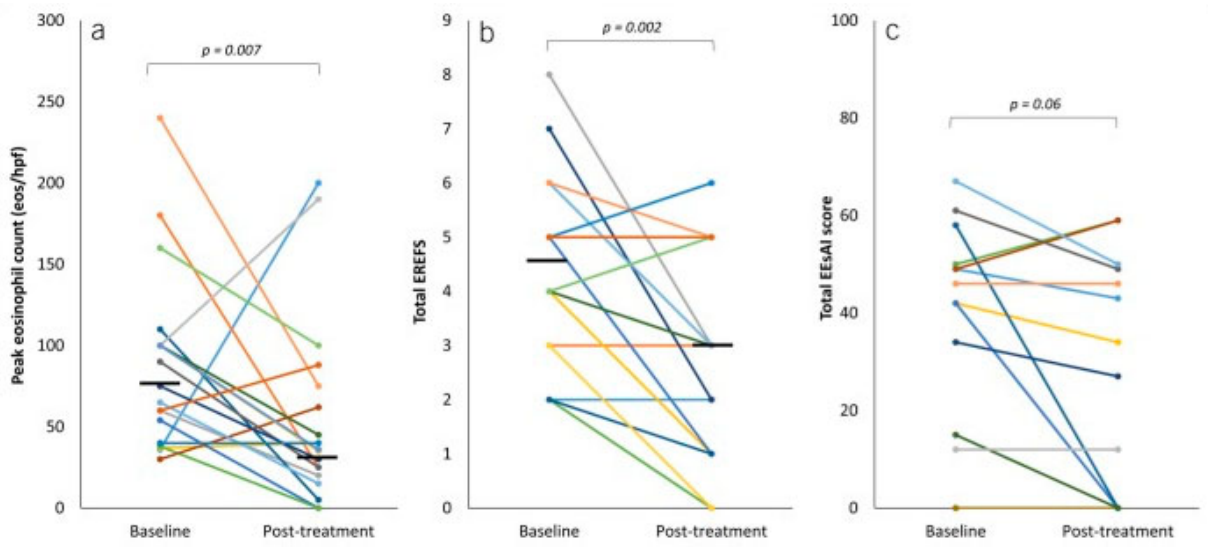
Future testing?

- Food specific IgG4 in biopsies



Wright BL et al. J Clin Allergy Immunol. 2016



Dellon ES et al. Clin Transl Gastroenterol 2019

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Nutrition

- «Six food elimination diet»
  - First retrospective study in pediatric population in 2006 by Kagalwalla et al
    - 39 children with SFED from 2003 – 2005, ~75% with ≤10 eos/hpf
  - Overcoming the insufficent sens/spec of allergy testing
  - More feasible in real life than elemental diet

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Six food elimination diet

Possibly β-casein A1 as culprit, A2 milk?

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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

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Combination?

- Potentially additive effect

- 4-FED + PPI vs. PPI mono
  - Eos <10/hpf in 88% vs. 45% after 12w
  - Reduction in peak Eos/hpf from 45 to 4

  Heine et al. J Allergy Clin Immunol. 2019
  Leung J et al. Gastro Hep Advances 2022

- Steroids + Elemental diet
  - 82% symptomatic improvement
  - slight reduction in furrows
  - non-significant reduction in Eos

  Reed CC et al. Clin Gastroenterol Hepatol. 2019 Dec

- Steroids + 2-FED
  - Reduction of median eos/hpf from 51 to 2
  - Improvement of symptoms

  Reed CC et al. Dig Dis Sci. 2018

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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 et al. *Allergy*. 2013

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Biologics

- Anti-IgE antibody (Omalizumab)
  - No effect

    Clayton F, Gastroenterology, 2014

- Anti-TNFα (Infliximab)
  - No effect

    Straumann A. J Allergy Clin Immunol. 2008 Aug

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Biologics

- Anti-IL 5 antibody (Mepolizumab and Reslizumab)
  - No improvement of symptoms
  - Significant reduction of eosinophilia (50-60%), but no histological remission

Straumann A et al. Gut. 2010
Spergel J.M. et al. J Allergy Clin Immunol. 2012

  - Trial for Mepolizumab finished 12/2022, results awaited

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Biologics

- IL-5Rα (Benralizumab, Fasenra©)
  - MESSINA phase III
  - Preliminary data from october with significant improvement of eos/hpf but not symptoms
  - Results to be presented

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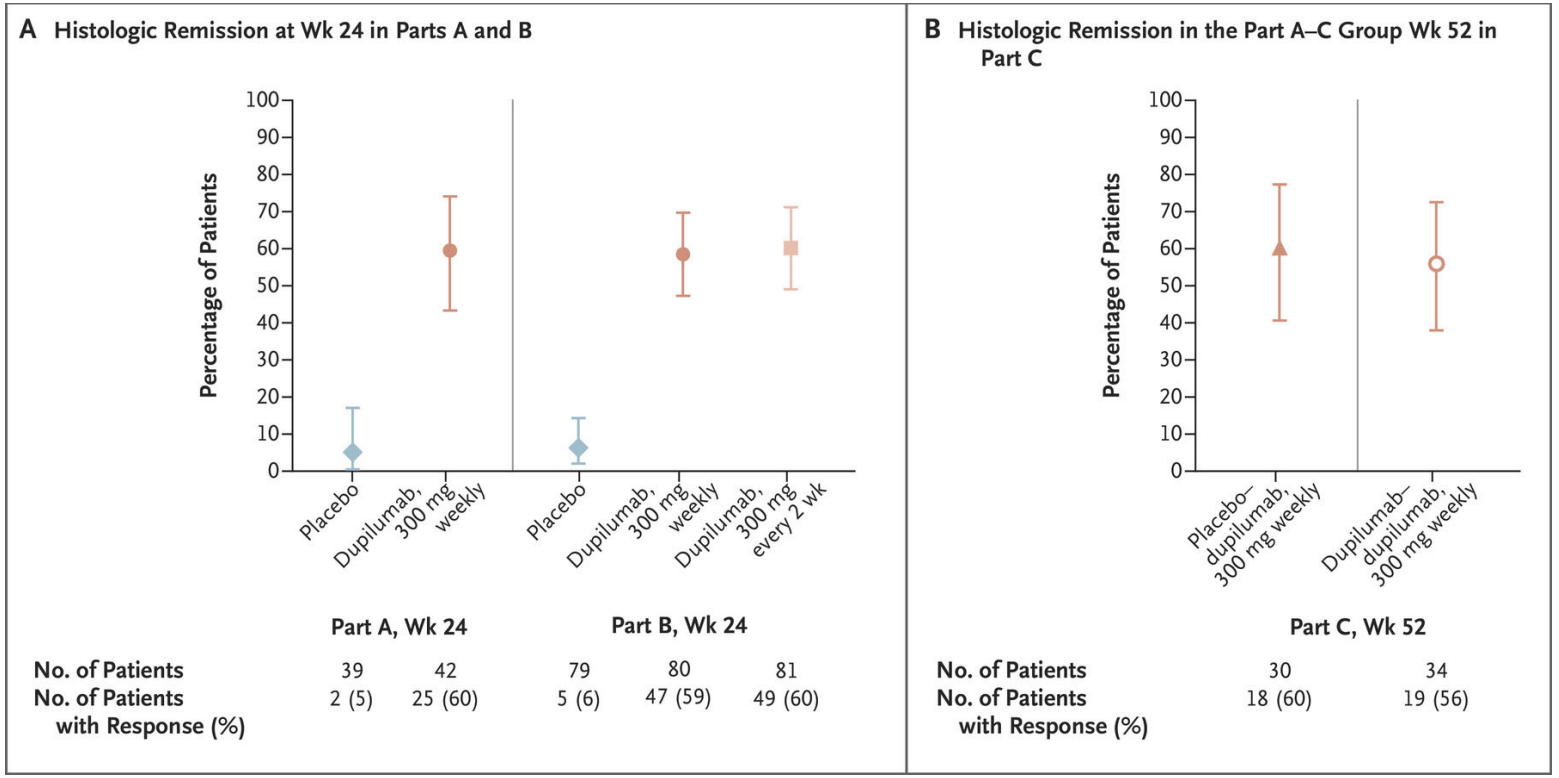
Dupilumab (IL-4/IL-13)



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Biologi

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  - Signif
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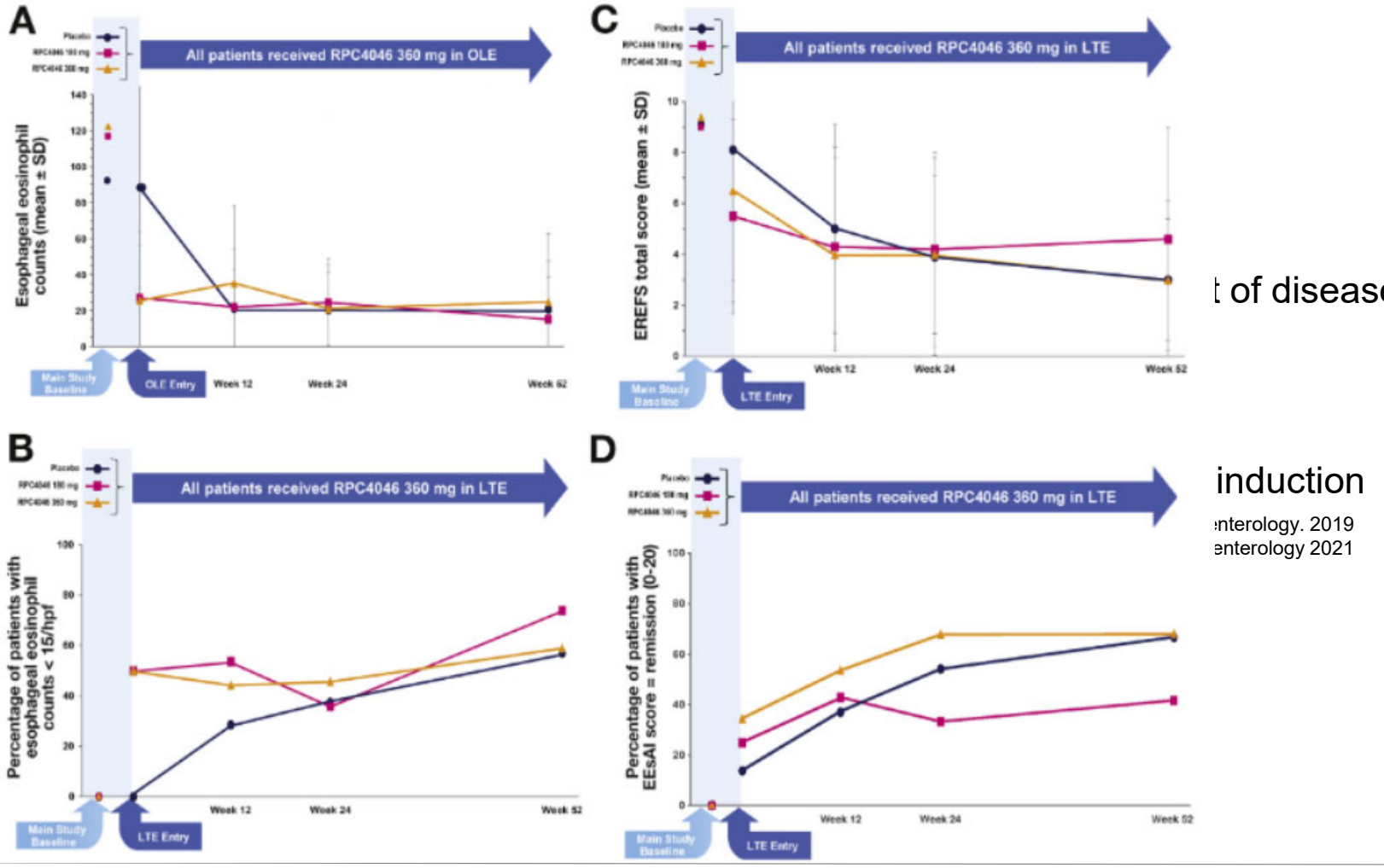

t of disease

induction

nterology. 2019
enterology 2021

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Awaited and ongoing trials

- Cendakimab
  - Phase III (NCT04753697, completion August 2024)
- Etrasimod (S1P receptor modulator)
  - Phase II double blind RCT (NCT04682639, completion July 2023)
- Lirentelimab (Sialic acid- binding immunoglobulintype lectin 8, Siglec-8)
  - Phase II/III multicenter double blind RCT (NCT04322708, completed May 2022)

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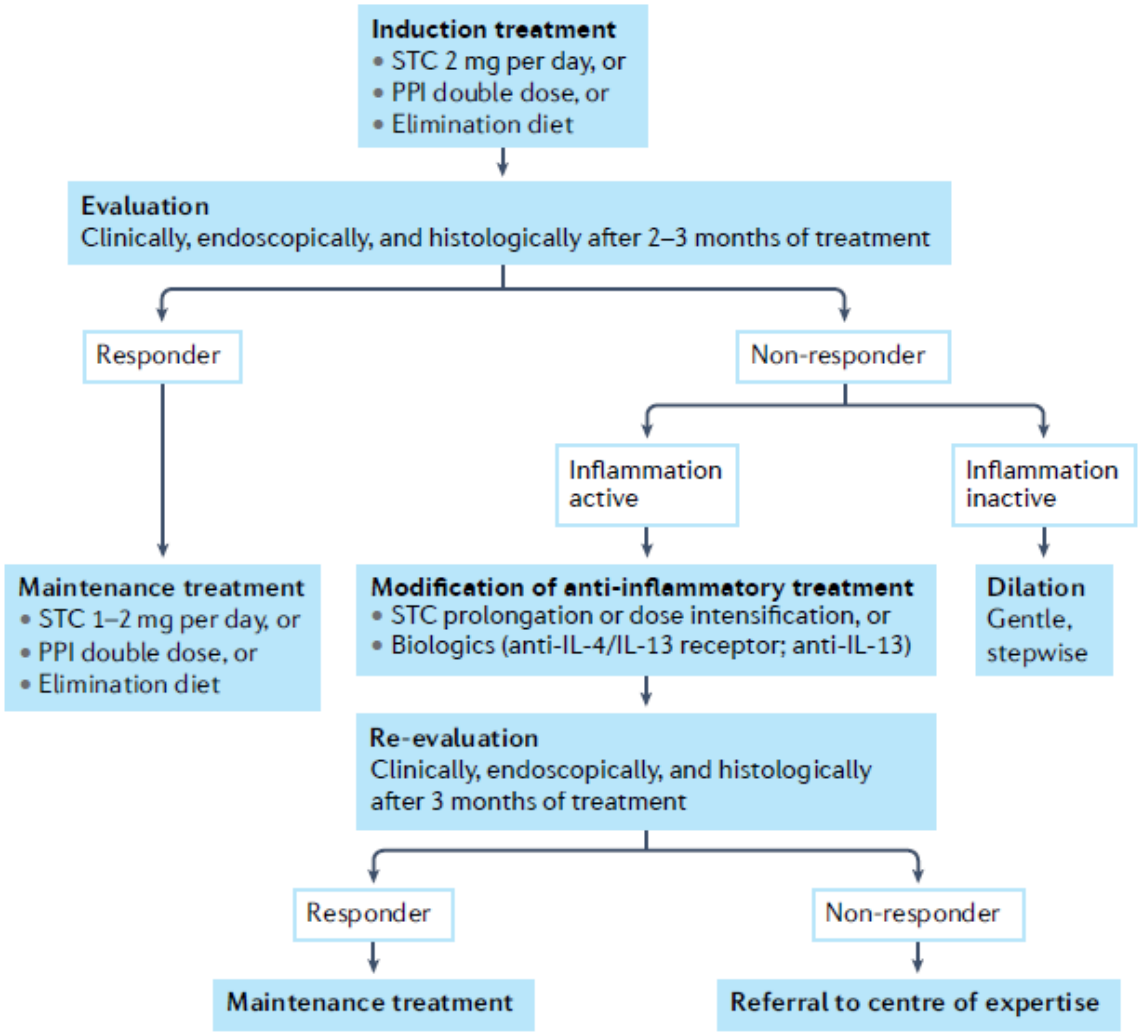
Treatm



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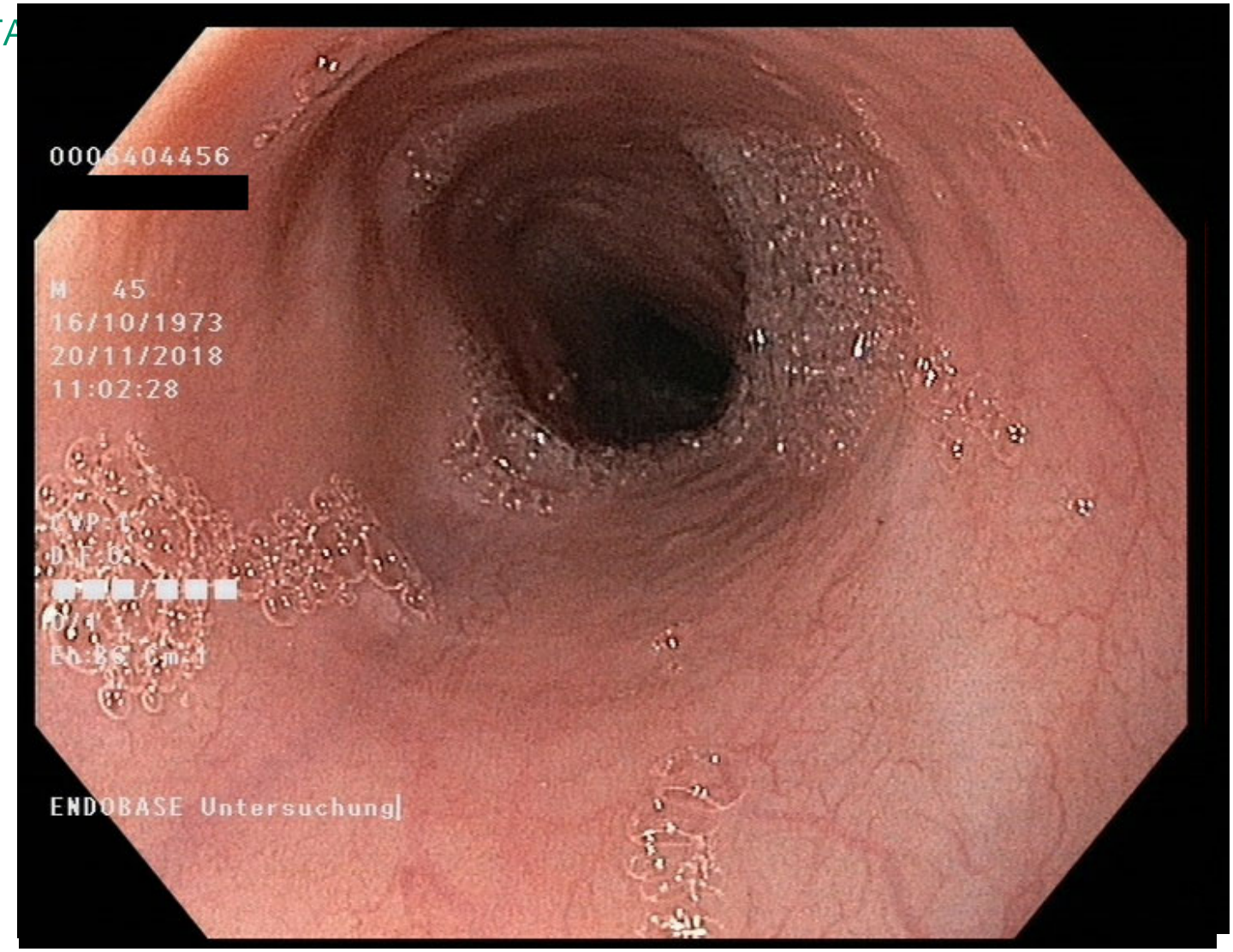
Strictures and dilatation

- Treatment of stenoses
  - Esophageal diameter <13mm, strictures
  - Mostly after anti-inflammatory treatment
  - Rarely first line -> impactations and daily dysphagia

  - Significantly reducing dysphagia, lasting up to 2 year
  - No increased risk for perforation (<1%)
  - Increase in post-procedural pain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ENDOBASE Untersuchung

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Monitoring

- Symptoms correlate insufficiently with inflammation
- Macroscopic-endoscopic aspect
- EREFS Score 88% Sens. / 92% Spec.

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 ~12w after treatment changes
- In stable disease at least 1x/year or possibly longer

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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
  - New biologics
- Regular control endoscopy for diagnosis and treatment of persistent inflammation or strictures

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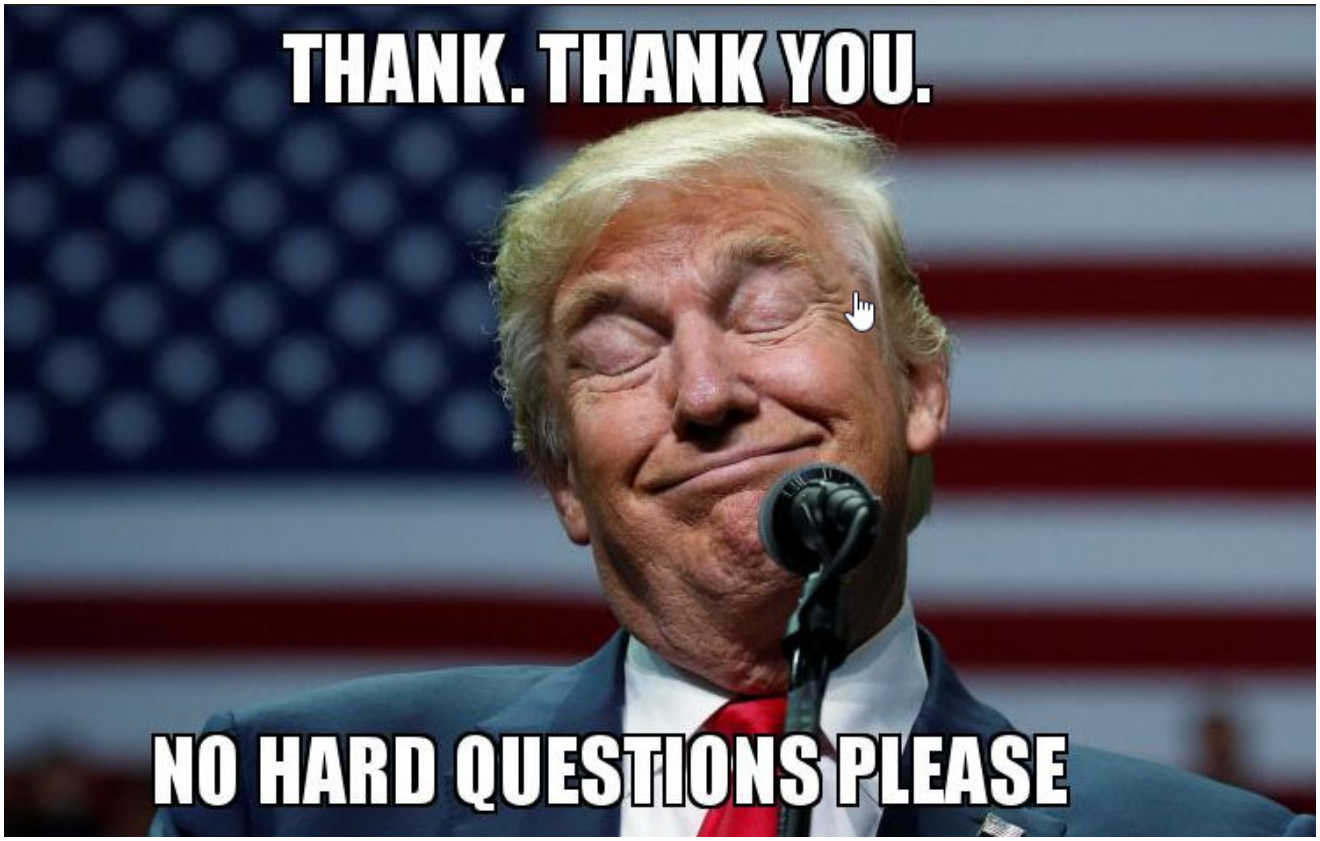

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Excursion eosinophilic gastroenteritis

- Epidemiology and pathogenesis similar to EoE

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

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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)