Esophageal epithelial permeability changes
in patients with allergic esophagitis.

No registrations found.

Ethical review	Positive opinion
Status	Recruiting
Health condition type	-
Study type	Interventional

Summary

ID

NL-OMON28827

Source
Nationaal Trial Register

Health condition

Eosinophilic esophagitis, pathophysiology, etiology, epithelial barrier integrity, permeability.
Eosinofiele oesofagitis, pathofysiologie, etiologie, epitheliale barrierefunctie, permeabiliteit.

Sponsors and support

Primary sponsor: Academic Medical Center, Amsterdam, The Netherlands
Source(s) of monetary or material Support: Netherlands Organisation for Scientific Research

Intervention

Outcome measures

Primary outcome

1. Size of esophageal epithelial intercellular spaces in EoE patients and healthy controls;

2. Permeability of esophageal mucosa to small molecules in EoE patients and healthy controls;

3. Tissue impedance of esophageal epithelium (in vivo) in EoE patients and healthy controls;

4. Numbers of esophageal intraepithelial mast cells and eosinophils in EoE patients and healthy controls.

Secondary outcome

Reversibility of abovementioned (permeability) changes by proton pump inhibition in EoE patients.

Study description

Background summary

The pathophysiology of EoE is largely unknown. We hypothesize that in EoE an impaired epithelial barrier due to acidic reflux could result in a deep penetration of food antigens into the epithelium and subsequent processing and activation of antigen presenting cells followed by activation of an inflammatory Th2 response.

Therefore, epithelial barrier function in EoE patients will be measured using several modalities and compared to epithelial barrier function of healthy controls. Furthermore, the effect of 8 weeks of proton pump inhibition on the epithelial barrier function will be determined in EoE patients.

Study objective

The pathophysiology of eosinophilic esophagitis (EoE) is largely unknown. We hypothesize that in EoE an impaired epithelial barrier due to acidic reflux could result in a deep penetration of food antigens into the epithelium and subsequent processing and activation of antigen presenting cells followed by activation of an inflammatory Th2 response. Evidence for an acid-induced impaired epithelial barrier in EoE will significantly contribute to our understanding of the pathophysiology of this disorder and therefore will be helpful for the development of an acceptable therapy.

Study design

All parameters are measured at baseline in EoE patients and healthy controls.

Measurements are repeated in EoE patients after 8 weeks of treatment with esomeprazole.

Intervention

Eosinophilic esophagitis patients are treated with esomeprazole 40 mg bd for 8 weeks.

The controls will receive no treatment.

Contacts

Public
Motility Center, room C2-231
Academic Medical Center
PO Box 22660
B.D. Rhijn, van
Amsterdam 1100 DD
The Netherlands
+31 (0)20 5665584
Scientific
Motility Center, room C2-231
Academic Medical Center
PO Box 22660
B.D. Rhijn, van
Amsterdam 1100 DD
The Netherlands
+31 (0)20 5665584

Eligibility criteria

Inclusion criteria

Eosinophilic esophagitis group:

1. Previous diagnosis of EoE confirmed by histopathology e.g. presence of >15 eosinophilic granulocytes per high power field (hpf) in mid-esophageal biopsies before the start of any therapy;

2. Written informed consent;

3. Age 18 – 75 years.

Healthy control group:

1. Written informed consent;

2. Age 18 – 75 years.

Exclusion criteria

Eosinophilic esophagitis group:

1. Inability to stop topical corticosteroids;

2. Inability to stop PPI, H2-receptor antagonist or prokinetic drug for 8 weeks;

3. Use of systemic corticosteroids, leukotriene inhibitors, or monoclonal antibodies, in the two month period preceding the study;

4. Use of anticoagulants;

5. Use of NSAIDs;

6. History of peptic ulcer disease;

7. History of Barrett’s esophagus;

8. History of GI cancer;

9. History of GI tract surgery (except appendectomy);

10. ASA class IV or V.

Healthy control group:

1. Use of systemic corticosteroids, leukotriene inhibitors, or monoclonal antibodies;

2. Use of anticoagulants;

3. Use of NSAIDs;

4. Personal history of atopic, skin or systemic diseases;

5. Symptoms suggestive of esophageal disease;

6. History of GI cancer;

7. History of GI tract surgery (except appendectomy);

8. History of PPI, H2-receptor antagonist, or prokinetic drug use;

9. ASA class IV or V.

Study design

Design

Study type:	Interventional
Intervention model:	Parallel
Allocation:	Non-randomized controlled trial
Masking:	Open (masking not used)
Control:	Active

Recruitment

NL

Recruitment status:	Recruiting
Start date (anticipated):	02-09-2011
Enrollment:	16
Type:	Anticipated

Ethics review

Positive opinion

Date:	06-06-2012
Application type:	First submission

Study registrations

Followed up by the following (possibly more current) registration

ID: 36166
Bron: ToetsingOnline
Titel:

Other (possibly less up-to-date) registrations in this register

No registrations found.

In other registers

Register	ID
NTR-new	NL3347
NTR-old	NTR3480
CCMO	NL36704.018.11
ISRCTN	ISRCTN wordt niet meer aangevraagd.
OMON	NL-OMON36166

Study results

Summary results
N/A