PWE-106

THE CAUSE OF DYSPHAGIA IN EOSINOPHILIC OESOPHAGITIS: OBSTRUCTION TO BOLUS PASSAGE NOT OESOPHAGEAL DYSMOTILITY

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Introduction Eosinophilic Oesophagitis (EO) presents with dysphagia and chest pain; however the cause of symptoms remains uncertain. Endoscopy reveals fibrotic and inflammatory mucosal disease but rarely tight stenosis. Conventional manometry with water swallows is usually normal; however this may not be clinically relevant as most patients report dysphagia on eating bread and meat but not on drinking liquids. This study applied HRM with water and solid bolus swallows to identify abnormal oesophageal function in EO patients and to associate abnormal pressure events with symptoms.

Methods Retrospective case review of 14 consecutive patients (10 male; age 36 (26–65)) on two sites referred for HRM with EO on biopsy of proximal and distal oesophagus. HRM studies

in the seated position included 10×5 ml water swallows and 5–10 solid (bread) swallows. 23 healthy volunteers (11 M:12 F, age 20–56) served as control. Association between abnormal pressure events and symptoms was assessed on a per patient and per swallow basis.

Results HRM identified oesophageal dysfunction in 3/14 (21%) patients with water swallows and 11/14 (79%) patients with solids ($p<0.008$). All 11 had increased intrabolus pressure gradient (IBPG) ≥30 mm Hg with solids (maximum at lower oesophageal sphincter (LOS) (n=9), mid-oesophagus (n=1) and upper oesophageal sphincter (n=1)).

Per patient: Typical symptoms were reported with IBPG >30 mm Hg by 1 (7%) patient with water and 7 (50%) patients with solids ($p=0.039$). Conversely, 7/11 (64%) patients with IBPG >30 mm Hg had symptoms (7/7 patients with IBPG >50 mm Hg).

Per swallow: There was temporal association between raised IBPG and patient reports of symptoms ($p<0.001$). Pan-oesophageal pressurisation >30 mm Hg triggered dysphagia; compartmentalised IBP >50 mm Hg between peristalsis and LOS triggered either dysphagia or chest pain. No association was present for any other pressure event.

Patients that had received steroids and/or dilation (n=7) had lower IBPG and reported less symptoms than untreated patients (n=7) (both $p<0.001$); however, if it occurred, the association between IBPG and symptoms remained.

One healthy subject had increased frequency of low-amplitude oesophageal spasm; however none had raised OGPG >30 mm Hg with solids and none reported symptoms.

Conclusion Patients with EO have normal motility but evidence of structural obstruction to solid bolus passage (usually across the LOS), presumably due to oesophageal stenosis or reduced LOS compliance due to fibrosis and inflammation. In all but one case this was evident only with solid bolus. Raised IBPG was closely associated with patient reports of symptoms and both improved on treatment.

Competing interests None.

Keywords Dysphagia, eosinophilic oesophagitis, Obstruction, oesophageal dysmotility, oesophageal pH and manometry studies.