When reviewing all 2WW referrals for gastroscopy the cancers pick up was 10% with the majority of examinations being normal or identifying insignificant findings.

**Conclusion** The two week wait referral system is often considered to be a poor method for detecting oesophagogastric cancer. In our data 10% patients referred in this manner had oesophagogastric cancer which is consistent with existing data. However when looking at all cases of of cancer diagnosed in this time period the 2WW represents the pathway for diagnosis for over half our malignancies (56%). Our cohort of patients showed similar TNM staging at the time of diagnosis irrespective whether they were referred routinely or on an urgent basis.

This suggests that the 2ww is an important pathway for referral of upper gastrointestinal malignancies but unfortunately does not identify patients at earlier stage. This is probably due to the lack of symptoms in early oesophagogastric cancer and strengthens the argument for identifying patients at an earlier stage perhaps by screening or surveillance of high risk groups.

**Disclosure of Interest** None Declared

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**Introduction**

Laparoscopic adjustable gastric banding (LAGB) is a common bariatric procedure in the UK due to its relative technical ease and reversibility. The technique has been around since the 1990s and although its immediate complications have been evident and known, the longer term complications are still emerging and not yet completely understood. Oesophageal dysmotility post-LAGB is now increasingly being recognised as a long-term complication associated with LAGB. This paper presents a potentially life-threatening complication associated with oesophageal dysmotility more than a decade after LAGB placement.

**Methods**

A 58yr old lady presented with chronic cough and mediastinal widening on chest X-ray. A computed tomogram (CT) revealed a mega-oesophagus with a collection in the mediastinum in keeping with a contained oesophageal perforation and a LAGB in situ. On further questioning, she mentioned that she had had a LAGB placed 12yrs previously. She had been experiencing recurrent coughs, chest infections, weight loss and dysphagia for 2 yrs but had not sought medical help.

**Results**

The LAGB was completely emptied (9mls of fluid). She was managed conservatively with nil orally, nasogastric drainage, antibiotics, parenteral nutrition over a period of 4 weeks and serial imaging was performed to monitor progress. She responded well to it, the perforation had completely healed, she resumed oral intake and was discharged.

**Conclusion**

While oesophageal dysmotility is emerging as a long-term complication occurring around 5–7 yrs post-LAGB, its association with oesophageal perforation has not been described in the literature prior to this incident. It is likely that oesophageal dysmotility resulted in mega-oesophagus and the associated reflux caused frequent coughing in our patient. The valsalva manoeuvre during coughing which closes the cricopharyngeus proximally and the presence of LAGB distally may have generated a high pressure zone within the oesophagus leading to perforation. This was a potentially life-threatening complication. This re-inforces the importance of life-long commitment to follow-up in patients who undergo bariatric surgery. We suggest at-risk patients developing mega-oesophagus should be identified and timely band–emptying performed to avoid this serious complication. Further long-term cohort studies need to be performed to determine the exact prevalence of oesophageal dysmotility and such complications.

**Disclosure of Interest** None Declared.

**A118**

**Disclosure of Interest** None Declared.
gastrointestinal pathologists who devote specific attention to identifying buried Barrett’s and buried dysplasia.

**Disclosure of Interest** None Declared

### PTU-172 C-MYC AS A BIOMARKER IN BARRETT’S OESOPHAGUS

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**Introduction** Identifying Barrett’s oesophagus (BE) patients at risk of progressing to oesophageal adenocarcinoma (EA) remains a challenge. Diagnosis of low grade dysplasia is limited by considerable intra and inter-observer variability and inflammation in biopsy samples can lead to a diagnosis of indefinite for dysplasia. Immunostaining using molecular biomarkers would therefore be useful as a diagnostic adjunct in the assessment of dysplasia.

The Aim of this study was to identify and validate a molecular biomarker that can objectively determine dysplasia status and thereby determine cancer risk in BE.

**Methods** Biomarkers of interest were identified through mining of a microarray gene expression dataset from 59 oesophageal samples with strict consensus diagnosis by expert pathologists [21 BE with no dysplasia (NDBE), 10 BE with low grade dysplasia (LGD), 13 BE with high grade dysplasia (HGD) and 8 EA]. Subsequent validation was performed on a BE tissue microarray (TMA) (60 NDBE, 19 LGD and 29 HGD), and EA TMA (n = 278).

**Results** Seventy eight genes were differentially expressed between NDBE and HGD. c-MYC was selected as a top target as the expression levels progressively increased with dysplasia stage and cancer. Sensitivity of the test (80–100%) is compromised in patients who have pancreatic exocrine insufficiency (PEI). Sensitivity is 73–100% for moderate to severe, but 0–63% for mild PEI. 1 Specificity of the test (80–100%) is compromised in patients who have other causes of diarrhoea or type 1 diabetes. 2,3,4 The aim of this observational study was to examine the causes of low faecal elastase in our hospital.

**Methods** A retrospective analysis of FE-1 tests performed between April 2010 and April 2012 was undertaken. The electronic medical notes of each patient with a low FE-1 (< 300 ug/l) were examined.

**Results** Of the 288 samples received by the laboratory, 23 patients had FE-1 of less than 100 ug/l (severe). 2 patients had FE-1 of 100–199 ug/l (moderate) and 19 patients had FE-1 of 200–299 ug/l (mild). The results were grouped as shown in Table 1. In 5 of the moderate PEI group there was insufficient information to determine the diagnosis. Non-pancreatic causes included microscopic colitis, coeliac disease, bile salt malabsorption, irritable bowel syndrome and infection.