Surprisingly in 53% of NDNE tissue samples we identified clonal expansion of cells (>10% mutant fraction) harbouring mutations in one or more of 13 of these putative driver genes. No difference in the frequency of mutation of these genes was observed between any of the disease stages studied. TP53 mutations clearly delineate between HGD/OAC and benign NDNE (p < 0.001). Whilst SMAD4 mutations are only observed in OAC (p < 0.001) demonstrating for the first time a clear genetic difference between the two.

Conclusion Mutagenic processes active in OAC are also active in the earliest stages of BE. Recurrent driver mutations identified in cancer may be acquired very early in the disease and may provide little or no progression advantage. Molecular diagnostic approaches must account for this.

Disclosure of Interest None Declared.

PTU-155 PROGRESSION OF LOW GRADE DYSPLASIA TO HIGH GRADE DYSPLASIA IN BARRETT’S OESOPHAGUS IN A SINGLE CENTRE


Introduction Barrett’s oesophagus is a common condition found in 4% of patients undergoing upper gastrointestinal endoscopy.1 The association between Barrett’s oesophagus and oesophageal adenocarcinoma has been well established. Scotland has a particularly high incidence of both Barrett’s and adenocarcinoma of the oesophagus.

The risk of progression from high grade dysplasia (HGD) to oesophageal adenocarcinoma (OAC) has been reported at approximately 10% per year.1 However the risk of progression from low grade dysplasia (LGD) is harder to quantify with studies showing progression to OAC from 0.6–1.69% per year, not dissimilar to that of non-dysplastic Barrett’s.2 More recently the SURF trail reported a 25% (17/68) progression from LGD to HGD/OAC in a surveillance group with LGD versus radiofrequency ablation.3

Methods In 2009 a clinical database of Barrett’s patients was developed in Forth Valley Hospital to ensure appropriate surveillance as per BSG guidelines. All patients diagnosed with Barrett’s were cared for by one responsible team, a dedicated Barrett’s endoscopy list was developed, and the use of narrow band imaging was introduced. At the end of 2012 the database was interrogated to assess the progress of all patients who had been diagnosed with LGD within the previous three years.

Results There were 915 patients with Barrett’s on the database, of which 829 were under follow up. 85 (10%) had LGD and of this patient cohort 19 had progressed to HGD. The progression rate from LGD to HGD was 22% (19/85). The median follow up of patients with LGD was 29 months (range 12–34 months). All patients who progressed from LGD to HGD had endoscopic therapy with endoscopic mucosal resection. On average 6 treatments and overall 3 sessions were performed in these patients per treatment in average 34 months. The progression rate from LGD to HGD was 22% (19/85). The median follow up of patients with LGD was 29 months (range 12–34 months). All patients who progressed from LGD to HGD had endoscopic therapy with endoscopic mucosal resection. On average 6 treatments and overall 3 sessions were performed in these patients per treatment in average 34 months. The progression rate from LGD to HGD was 22% (19/85). The median follow up of patients with LGD was 29 months (range 12–34 months). All patients who progressed from LGD to HGD had endoscopic therapy with endoscopic mucosal resection. On average 6 treatments and overall 3 sessions were performed in these patients per treatment in average 34 months. The progression rate from LGD to HGD was 22% (19/85). The median follow up of patients with LGD was 29 months (range 12–34 months). All patients who progressed from LGD to HGD had endoscopic therapy with endoscopic mucosal resection. On average 6 treatments and overall 3 sessions were performed in these patients per treatment in average 34 months. The progression rate from LGD to HGD was 22% (19/85). The median follow up of patients with LGD was 29 months (range 12–34 months). All patients who progressed from LGD to HGD had endoscopic therapy with endoscopic mucosal resection. On average 6 treatments and overall 3 sessions were performed in these patients per treatment in average 34 months.

Conclusion The progression rate from LGD to HGD is similar to reported rates found in the SURF trial. This suggests that LGD carries a greater risk of progression, and therefore worse prognosis than previously reported. This is a potential group of patients in whom to consider early intervention rather than adopt the standard surveillance strategy. Further studies to evaluate the effectiveness of treatment rather than surveillance in this group should be considered.

REFERENCES