Supplementary table 1. Comprehensive summary of tissue biomarker studies to predict prevalent dysplasia and malignant potential in Barrett's oesophagus

Biomarker	Study	Finding	Technique for identification	Sample size	EDRN stage	Grade of evidence
Abnormal DNA content (Tetraploidy, Aneuploidy)	Reid et al 2000 (128)	28% 5-year cumulative OAC incidence compared to 0% in absence	Flow cytometry on frozen biopsies	322	Phase 3/4: prospectively collected samples retrospective analysis	III
	Reid et al 2001 (129)	RR 7.5 p<0.001 CI of progression to OAC	Flow cytometry on frozen biopsies	325	Phase 3/4: prospectively collected samples retrospective analysis	III
	Dunn et al 2010 (130)	End point HGD/OAC. Hazard ratio 8.2 (1.8-37.8, P=0.001)	Image cytometry on paraffin embedded biopsies	30 (patients without dysplasia following PDT for dysplasia)	Phase 4	III
	Galipeau et al 2007 (131)	Cancer end point. At 10 years RR=8.5 (95% CI 4.3-17.0)	Flow cytometry on frozen biopsies	243	Phase 3/4: prospectively collected samples retrospective analysis	III
	Fritcher et al 2008 (132)	All patients with a polysomic FISH result had HGD / OAC within 6 months. There was a significant difference over time between FISH diagnostic categories for progression to HGD/adenocarcinoma (*P* < .001)	FISH on endoscopic brushings	97 patients, 84 of whom had a biopsy-confirmed HGD/OAC	Prospective Phase 4	III
	SIkkema et al 2009 (133)	HR 3.5;(95% CI: 1.3-9.4) Did not remain a risk factor on multivariable analysis	Flow cytometry on frozen biopsies	Progressors n=27, non-progressors n=27	Prospective phase 4	IIa
(17p)p53 LOH	Reid et al 2001 (129)	RR =16, p<0.001	Flow cytometry on frozen biopsies	325	Phase 3/4: prospectively collected samples retrospective analysis	III
	Galipeau et al 2007 (131)	RR=10.6 (95% CI 5.2-21.3, p<0.001)	Flow cytometry on frozen biopsies	243	Phase 3/4: prospectively collected samples retrospective analysis	III
p53 positive on IHC	Weston et al 2001 (134)	Kaplan-Meier curves differed significantly between p53 positive and negative patients for outcome defined as progression of LGD	IHC	Progressors n=5, non-progressors n=43	Prospective phase 4	IIa
	Murray et al 2006 (135)	OAC/HGD endpoint: OR 8.42 (95% CI 2.37-30.0)	IHC	Progressors n=35, controls n=175	Phase 3: Retrospective	IIa
	SIkkema et al 2009 (133)	HR 6.5 (95% CI: 2.5-17.1) Remained a risk factor on multivariable analysis	IHC	Progressors n=27, non-progressors n=27	Prospective phase 4	IIa
	Younes et al 1997 (136)	Progression from LGD to HGD/OAC. P = 0.0108. p53 accumulation has a sensitivity of 100%, specificity of 93%, and a predictive value of a positive test of 0.56	IHC	Progressors n=5, Non-progressors n=25	Phase 3: Retrospective	IIa
	Skacel et al 2002 (126)	Progression from LGD to HGD/OAC. A correlation with clinical progression was seen p = 0.017. (88% sensitivity and 75% specificity for progression)	IHC	Progressors n=8, Non-progressors n=8	Phase 3: Retrospective	IIa
	Bani_Hani 2000 (137)	OR = 2.99 (95% CI = 0.57-15.76; P =0.197).	IHC	Nested case control (unmatched) n=12 cases	Phase 3: Retrospective	IIa
	Kastelein 2012 (127)	RR = 6.2 (95% CI = 3.6 – 10.9)	IHC	Progressors n=49 Non-progressors	Phase 3: Retrospective	IIa

				n=586		
9p (p16) LOH	Galipeau et al 2007 (131)	RR=2.6 (95% CI 1.1-6.0, p=0.03).	Flow cytometry on frozen biopsies	243	Phase 3/4: prospectively collected samples retrospective analysis	III
Mcm2	Sirieix et al 2003(138)	Progressors had higher Mcm2 expression prior to the development of dysplasia than matched controls (mean, 28.4 and 3.4% positive cells, respectively, P < 0.0001).	IHC	Cases n=9, controls n+18	Phase 3: Retrospective	IIa
Cyclin A	Lao-Sirieix et al 2007 (139)	OR 7.5 (95% CI 1.8-30.7)	IHC	Nested case control; n=16 cases, n= 32 controls.	Phase 3: Retrospective	IIa
Cyclin D	Bani_Hani 2000(137)	OR = 6. 85; 95% CI = 1.57-29.91; P =.0106)	IHC	Nested case control (unmatched) n=12 cases	Phase 3: Retrospective	IIa
Methylation	Schulmann et al 2005 (140)	Hypermethylation of p16 (OR= 1.74, 95% CI 1.33-2.20), RUNX3 (OR 1.80, 95% CI 1.08-2.81), and HPP1 (OR 1.77, 95% CI 1.06-2.81) were independently associated with risk of progression	Real-time quantitative methylation-specific PCR	Progressors n=8, non progressors n=45	Phase 3: Retrospective longitudinal	IIa
	Jin et al 2009(141)	With specificity at 0.9, sensitivities of progression prediction approached 50% based on a panel of 8 methylation biomarkers	Real-time quantitative methylation-specific PCR	Progressors n=50, non-progressors n=145	Phase 3: Retrospective	IIa
	Wang et al 2009(142)	Progressors to HGD/OAC had higher prevalence of p16 hypermethylation in their index biopsy compared with those who did not progress (100 vs. 33%; P=0.008)		Progressors n=7, non-progressors n=50	Phase 3: Retrospective	IIa
Clonal diversity	Merlo et al 2010(143)	All diversity measures were strong and highly significant predictors of progression (Cox proportional hazards model, P< 0.001).	Fresh frozen biopsies purified from non-proliferating stroma, DNA extraction, amplification, genotyping and FACS	Progressors to OAC n=33, non-progressors n=206	Phase 3/4: Prospectively collected samples retrospective analysis	IIa
Combination panels	Galipeau et al 2007(131)	17p LOH, 9p LOH + DNA content abnormality combination. RR 38.7 (95% CI 10.8-138.5, p<0.001)	Flow cytometry, DNA extraction and whole-genome amplification on frozen biopsies	243	Phase 3/4: Prospectively collected samples retrospective analysis	III
	Bird-Lieberman et al 2012 (114)	LGD, DNA ploidy abnormality and AOL	Histology, Image cytometry and IHC	Progressors to OAC n=89, non-progressors n=291	Phase 3: Retrospective population-based, nested case-control	III

Confidence interval (CI), relative risk (RR), photodynamic therapy (PDT), fluorescence in-situ hybridisation (FISH), immunohistochemistry (IHC), loss of heterozygosity (LOH), Hazard ratio (HR), odds ratio (OR)

Supplementary table 2. Summary of cost-effectiveness models in Barrett's oesophagus

Study	Comparison	Population	Outcome	Annual cancer conversion rate	Uncertainty evaluated	Time	Discount (annual rate)	Perspective
Inadomiet al, 2003	Screening and surveillance versus do nothing	50 year old white male population with chronic GERD	$12,336/QALY for 5 year surveillance	0.5%	One way sensitivity analysis	30 years	3% costs and benefits	Third party payer, US
Gersonet al, 2004	Screening and surveillance versus do nothing	50 year old male population with chronic GERD	$12,140 per life year gained for surveillance	0.5%	One way sensitivity analyses	Life time	3% costs and benefits	Not explicitly stated
Gupta et al, 2011	Screening and surveillance versus do nothing	50 year old patient attending for colonoscopy screening	$95,559/QALY gained for surveillance	0.5%	One way sensitivity analysis	30 years	3% costs and benefits	Third party payer, US
Nietert et al, 2003	Screening and surveillance versus do nothing	50 year old person with chronic GERD	$86,833/QALY gained for surveillance	0.44%	One way sensitivity analysis	Life time	3% costs and benefits	Third party payer, US
Inadomi et al, 2009	Ablation versus do nothing	50 year old person with non-dysplastic Barrett's	$16,286/QALY for ablation	0.5%	PSA, Ablation >80% chance cost-effective at WTP $100,000	30 years	3% costs and benefits	Third party payer, US
Das et al, 2009	Surveillance every 3 years versus do nothing	Male aged 50 with non dysplastic Barrett's	$86,434/QALY gained for surveillance	0.5%	PSA, approx. 60% chance that surveillance is cost-effective	Life time	3% costs and benefits	Societal perspective, US
Provenzale et al, 1999	Surveillance every 1-5 years versus do nothing	55 year old patients with non dysplastic Barrett's	$98,000/QALY for 5 year surveillance	0.44%	One way sensitivity analysis	Life time	5% costs and benefits	Third party payer, US
Somerville et al, 2008	Surveillance every 3 years versus do nothing	Male aged 55 with non dysplastic Barrett's	Do nothing dominates	unclear	PSA, "substantial uncertainty" approximately a 15% chance surveillance is cost effective at a WTP of pounds 50,000/QALY	20 years	Costs discounted 6%, benefits discounted 1.5%	Third party payer, UK
Sonnenberg et al, 2002	Surveillance every 2 years versus do nothing	60 year old patients with non dysplastic Barrett's	$16,695 for surveillance	0.5%	Multivariate sensitivity analysis	unclear	3% costs and benefits	Third party payer, US
Benaglia et al. 2013	No screening vs endoscopy screening vs Cytosponge screening	50 years old men with GORD symptoms	$ 22,167 / QALY for endotherapy $ 15,724 / QALY for Cytosponge	Transition matrix between all Barrett's states	PSA and deterministic sensitivity analysis	Up to intervening death (up to 50 years)	3% costs and benefits	NHS, UK
Inadomi et al, 2009	Ablation versus oesophagectomy versus surveillance	50 year old person with HGD	RFA dominant	5.5% for HGD, 64% efficacy of RFA	PSA, different ablation techniques may be cost effective but all almost 100% chance of being cost effective at WTP $50,000	30 years	3% costs and benefits	Third party payer, US
Hur et al,	PDT versus	Male aged	PDT	15% (first	One way	Life	3%	Societal

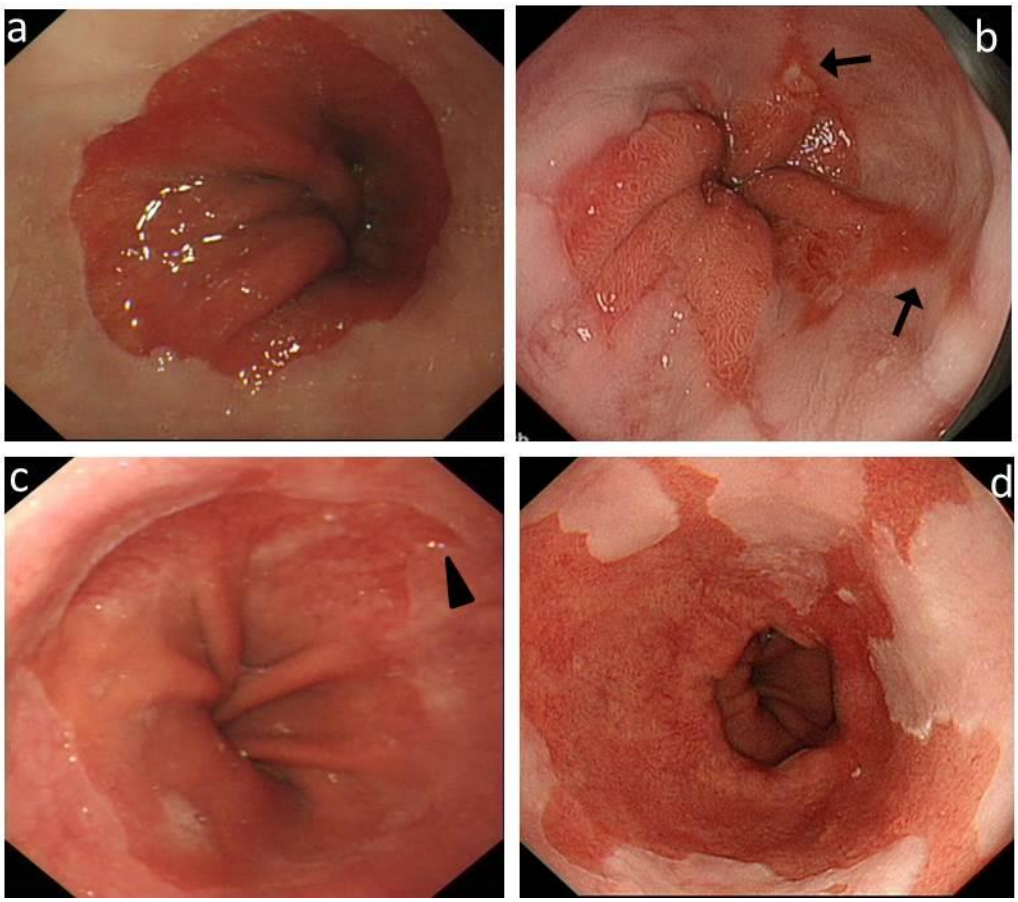
2003	oesophagectomy versus surveillance every 2 years	55 with HGD	$12,400/QALY versus surveillance and $3,300/QALY versus surgery	year) for surveillance, 6.5% for PDT and 1.6% for surgery	sensitivity analysis	time	costs and benefits	perspective, US
Das et al, 2009	RFA versus surveillance	Male aged 50 with non dysplastic Barrett's	RFA dominant	0.5% for surveillance, 0.25% for RFA	PSA, approx. 70% chance that RFA cost-effective	Life time	3% costs and benefits	Societal perspective, US
Comay et al, 2007	PDT versus oesophagectomy versus surveillance	Male patients aged 50 with HGD	$879/QALY for PDT	20.8% for HGD, 6.8% after RFA, 0% after surgery	PSA, RFA >99% chance being cost-effective at WTP $50,000/QALY	5 years	3% costs and benefits	Third party payer, Canadian
Vij et al, 2004	PDT versus oesophagectomy versus surveillance	Male patients aged 55 with HGD	$47,410/QALY for PDT	30% for HGD, 7% for PDT, 0% for surgery	One way sensitivity analysis	Life time	3% costs and benefits	Third party payer, US
Boger et al, 2010	RFA versus oesophagectomy	Male patients aged 64 with HGD	RFA dominant	1.4% after RFA, 0.2% after surgery	PSA, RFA >85% chance cost effective	5 years	3.5% costs and benefits	Third party payer, UK

Appendix 1. Percentages of agreement on statements and rounds of voting required for approval of individual statements

Section	Statement	Agreement					Rounds
Diagnosis	Barrett's oesophagus is defined as an oesophagus in which ...	A+ 64%	A 26%	U 0%	D 5%	D+ 5%	1
	The proximal limit of the longitudinal gastric folds with ...	A+ 64%	A 26%	U 10%	D 0%	D+ 0%	1
	Endoscopic reporting should be done using a minimum ...	A+ 95%	A 5%	U 0%	D 0%	D+ 0%	1
	In order to improve the standard of care and to ease ...	A+ 42%	A 48%	U 10%	D 0%	D+ 0%	1
Screening	Screening with endoscopy is not feasible or justified ...	A+ 53%	A 42%	U 0%	D 0%	D+ 5%	1
	Endoscopic screening can be considered in patients ...	A+ 41%	A 41%	U 18%	D 0%	D+ 0%	2
Surveillance	Although randomised controlled trial data are lacking ...	A+ 26%	A 69%	U 5%	D 0%	D+ 0%	1
	Endoscopic monitoring with histopathological assessment ...	A+ 26%	A 69%	U 5%	D 0%	D+ 0%	1
	Surveillance regimens should take into account ...	A+ 61%	A 39%	U 0%	D 0%	D+ 0%	2
	Dysplasia confirmed by two GI pathologists ...	A+ 72%	A 22%	U 0%	D 6%	D+ 0%	1
	Until randomised controlled evidence is available ...	A+ 72%	A 22%	U 0%	D 6%	D+ 0%	1
Practicalities of endoscopic surveillance	Patients should have early access to an outpatient clinic ...	A+ 69%	A 26%	U 5%	D 0%	D+ 0%	1
	For a given patient whether or not surveillance is indicated ...	A+ 53%	A 37%	U 5%	D 5%	D+ 0%	1
	High-resolution endoscopy should be used in Barrett's ...	A+ 32%	A 53%	U 10%	D 5%	D+ 0%	1
	Standard trans-oral endoscopy should be preferred ...	A+ 48%	A 37%	U 15%	D 0%	D+ 0%	1
	There is not sufficient evidence to recommend routine use ...	A+ 34%	A 60%	U 6%	D 0%	D+ 0%	1
	Adherence to a quadrantic, 2cm biopsy protocol ...	A+ 59%	A 41%	U 0%	D 0%	D+ 0%	1
	Surveillance is generally not recommended in patients ...	A+ 47%	A 47%	U 6%	D 0%	D+ 0%	1
	For patients with Barrett's oesophagus shorter than 3 cm ...	A+ 17%	A 72%	U 11%	D 0%	D+ 0%	2
	Patients with Barrett's oesophagus shorter than 3 cm, with ...	A+ 35%	A 53%	U 6%	D 6%	D+ 0%	2
	Patients with segments of 3 cm or longer should ...	A+ 39%	A 49%	U 0%	D 6%	D+ 6%	2
Histopathological diagnosis of dysplasia	Given the important management implications for a ...	A+ 69%	A 26%	U 5%	D 0%	D+ 0%	1
	Given the difficulties associated with the management of ...	A+ 69%	A 26%	U 5%	D 0%	D+ 0%	1
	The addition of a p53 immunostaining to the histopath- ...	A+ 32%	A 58%	U 5%	D 5%	D+ 0%	1
Management of Dysplasia and Early Cancer	Patients with a diagnosis of indefinite for dysplasia ...	A+ 26%	A 64%	U 10%	D 0%	D+ 0%	1
	Management of low grade dysplasia (LGD) is unclear ...	A+ 61%	A 39%	U 0%	D 0%	D+ 0%	2
	Expert high resolution endoscopy (HRE) should be carried ...	A+ 59%	A 36%	U 5%	D 0%	D+ 0%	1
	Visible lesions should be considered malignant until ...	A+ 59%	A 36%	U 5%	D 0%	D+ 0%	1
	Description of lesion morphology using the Paris classif- ...	A+ 42%	A 42%	U 16%	D 0%	D+ 0%	1
	All patients with dysplasia or early cancer, for whom ...	A+ 59%	A 41%	U 0%	D 0%	D+ 0%	1
	Patients with dysplasia or early cancer should be ...	A+ 59%	A 41%	U 0%	D 0%	D+ 0%	1
Endoscopic therapy for Barrett's related neoplasia	For high grade dysplasia and Barrett's-related adenoca- ...	A+ 53%	A 37%	U 10%	D 0%	D+ 0%	1
	Endoscopic therapy of Barrett's neoplasia should be ...	A+ 72%	A 17%	U 11%	D 0%	D+ 0%	2
	A minimum of 30 supervised cases of Endoscopic Res- ...	A+ 50%	A 39%	U 11%	D 0%	D+ 0%	2
	Endoscopic Resection should be performed in high ...	A+ 33%	A 62%	U 5%	D 0%	D+ 0%	3
ER for Barrett's related	Endoscopic assessment will usually identify the area ...	A+ 63%	A 37%	U 0%	D 0%	D+ 0%	1

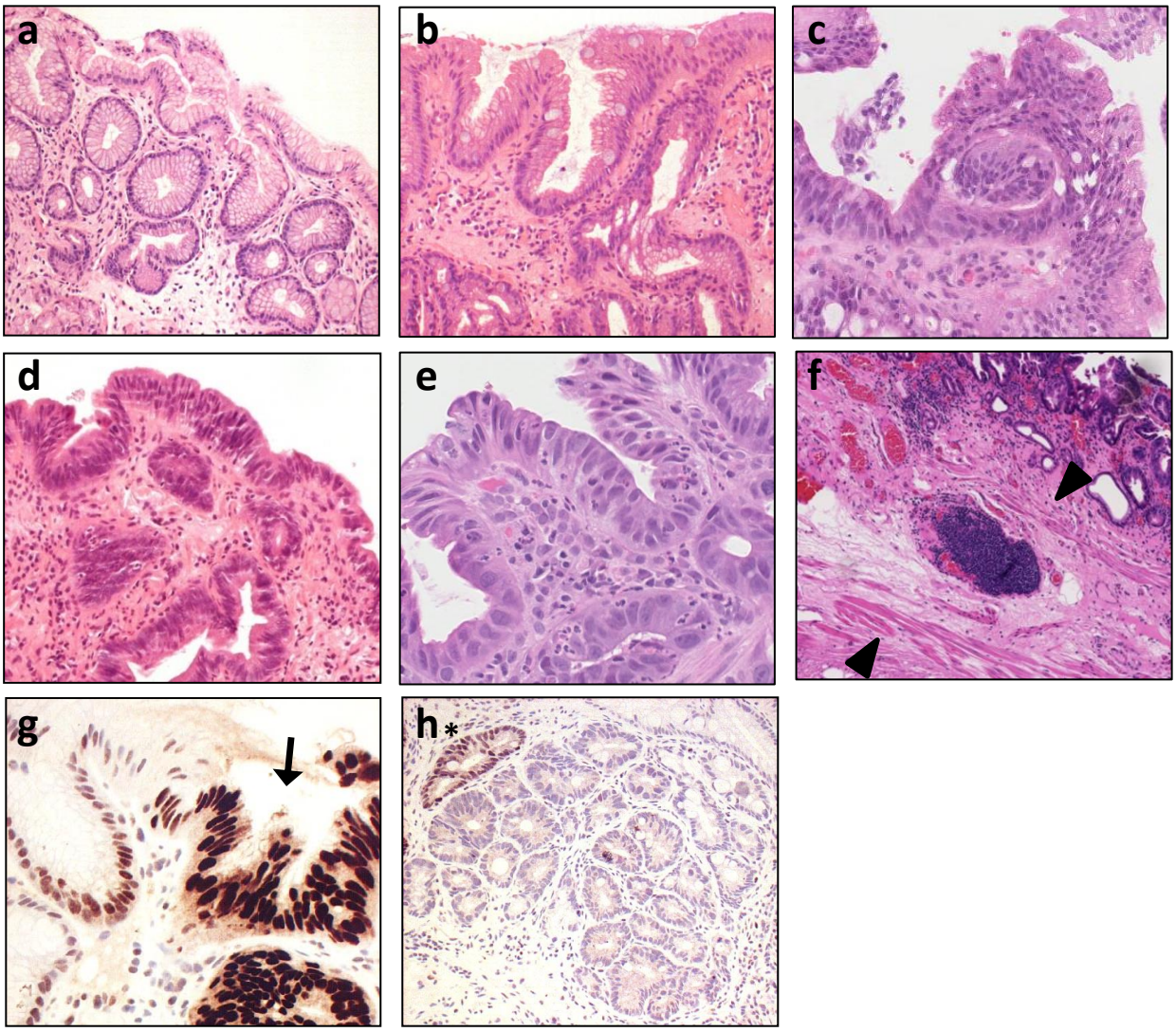
neoplasia associated with visible lesions	ER is recommended as the most accurate staging interv- ...	A+ 59%	A 36%	U 5%	D 0%	D+ 0%	1
	ER should be considered the therapy of choice for dyspl- ...	A+ 59%	A 36%	U 5%	D 0%	D+ 0%	1
	For patients at high surgical risk endoscopic therapy ...	A+ 59%	A 36%	U 5%	D 0%	D+ 0%	1
	For T1b adenocarcinomas with involvement of the second ...	A+ 59%	A 36%	U 5%	D 0%	D+ 0%	1
	The cap and snare technique with sub-mucosal injection ...	A+ 48%	A 52%	U 0%	D 0%	D+ 0%	1
Pathology reporting of ER	Use of a minimum data set for the reporting of endosc- ...	A+ 63%	A 32%	U 5%	D 0%	D+ 0%	1
	The presence of tumour cells at deep margin indicates ...	A+ 63%	A 32%	U 5%	D 0%	D+ 0%	1
Imaging for HGD and T1 carcinoma	Neither CT nor PET-CT have a clear role in the staging ...	A+ 48%	A 42%	U 10%	D 0%	D+ 0%	1
	CT and PET-CT should be performed in cases with subm- ...	A+ 48%	A 42%	U 10%	D 0%	D+ 0%	1
	Since EUS can both overstage and understage T1 lesions ...	A+ 32%	A 63%	U 5%	D 0%	D+ 0%	1
	In selected cases where the endoscopist cannot exclude ...	A+ 33%	A 56%	U 11%	D 0%	D+ 0%	1
	EUS +/- FNA of visible lymph nodes is recommended in sel- ...	A+ 32%	A 63%	U 5%	D 0%	D+ 0%	1
Ablative therapy for flat HGD and residual Barrett's post-ER	In the presence of HGD without visible lesions (flat HGD) ...	A+ 68%	A 32%	U 0%	D 0%	D+ 0%	1
	Eradication of residual Barrett's oesophagus after focal ...	A+ 74%	A 21%	U 5%	D 0%	D+ 0%	1
	Endoscopic follow up is recommended following ...	A+ 53%	A 42%	U 5%	D 0%	D+ 0%	1
Surgical management of early Barrett's neoplasia	Surgical therapy is considered the treatment of choice for ...	A+ 59%	A 41%	U 0%	D 0%	D+ 0%	1
	Oesophagectomy should be performed in high volume ...	A+ 59%	A 41%	U 0%	D 0%	D+ 0%	1
	There is currently no evidence to support one technique of ...	A+ 53%	A 47%	U 0%	D 0%	D+ 0%	1
	There is not sufficient data to recommend endoscopic ...	A+ 32%	A 53%	U 15%	D 0%	D+ 0%	1
Audit for HGD and early cancer	Findings and management decisions for HGD and early ca ...	A+ 47%	A 47%	U 6%	D 0%	D+ 0%	1
Economic considerations	There are insufficient data to indicate that endoscopic ...	A+ 55%	A 39%	U 6%	D 0%	D+ 0%	2
	Endoscopic therapy for dysplastic Barrett's oesophagus ...	A+ 77%	A 23%	U 0%	D 0%	D+ 0%	2
Chemoprevention and symptom control	There is not yet sufficient evidence to advocate acid suppr- ...	A+ 42%	A 53%	U 5%	D 0%	D+ 0%	1
	Use of medication to suppress gastric acid production ...	A+ 42%	A 53%	U 5%	D 0%	D+ 0%	1
	PPIs have the best clinical profile for symptomatic manag- ...	A+ 42%	A 53%	U 5%	D 0%	D+ 0%	1
	There is not sufficient evidence to recommend anti-reflux ...	A+ 59%	A 31%	U 5%	D 0%	D+ 5%	1
	Anti-reflux surgery should be considered in patients with ...	A+ 59%	A 31%	U 5%	D 0%	D+ 5%	1
	There is currently insufficient evidence to support the use ...	A+ 53%	A 37%	U 10%	D 0%	D+ 0%	1
Patient perspective	All patients should be offered an appointment to discuss ...	A+ 59%	A 41%	U 0%	D 0%	D+ 0%	1
Future developments	The following developments would revolutionise the care ...	A+ 37%	A 48%	U 5%	D 10%	D+ 0%	1

Appendix 2. Endoscopic examples of normal GOJ, irregular Z-line and Barrett's oesophagus



a. Normal GOJ with a squamo-columnar junction which coincides with the top of the gastric folds.
b. Irregular Z-line (arrows indicate focal areas of oesophagitis, which can mimic tongues of Barrett's oesophagus)
c. Irregular Z-line (arrow head shows a tongue of columnar-lined oesophagus shorter than 1 cm, which does not fulfill the minimum length required for an endoscopic diagnosis of Barrett's oesophagus)
d. Clearly visible Barrett's oesophagus on endoscopic imaging.

Appendix 3. Histopathological and immunohistochemical pictures



a. Barrett's oesophagus with gastric metaplasia only
b. Barrett's oesophagus with intestinal metaplasia
c. Barrett's oesophagus with indefinite for dysplasia
d. Barrett's oesophagus with LGD
e. Barrett's oesophagus with HGD
f. Duplicated muscularis mucosae (arrowheads show the two layers of muscularis mucosae with the stroma in between)
g. Example of significant p53 staining pattern (arrow shows glands with p53 overexpression compared to adjacent glands on the left)
h. Another example of significant p53 staining pattern (loss of p53 staining in the majority of the Barrett's glands compared to background staining in an adjacent gland pointed by the arrow)

Appendix 4. Information for patients with Barrett's oesophagus

What is Barrett's oesophagus?
Barrett's oesophagus is a change in the cells lining the gullet to a different cell type not normally found in this organ. It tends to occur in people suffering from acid and bile reflux, which often causes heartburn and indigestion symptoms. It is also more frequent in people with a hiatus hernia, which is an impairment of the valve that normally prevents acid juices passing from the stomach to the gullet. Men are more frequently affected than women, although it can affect people of either sex and at any age.

Can Barrett's oesophagus lead to cancer and what monitoring is required?
There is a connection between Barrett's oesophagus and a type of cancer of the gullet, called oesophageal adenocarcinoma. Although the majority of patients with Barrett's will never develop cancer, a rough estimate is that approximately 7% of people with Barrett's may go on to develop cancer during their lifetime. Because of this, it is recommended that patients with Barrett's oesophagus are monitored with an endoscopy (camera test) in order to detect any cancer occurring at a very early and curable stage. During this test, the doctor also takes multiple small tissue samples (biopsies) to be examined under the microscope for cellular changes.
For some patients, the risk of cancer is extremely small. For example patients with a very short Barrett's (1 or 2 cm) have a very small risk and therefore may not need repeat endoscopy or require one endoscopy every 3 or 5 years depending on the cell types present in the biopsy. Some other patients with longer segments and the cell type called intestinal metaplasia have a slightly higher risk and may require and endoscopy every 2 or 3 years. If cellular changes called dysplasia are found under the microscope, it may be recommended to have an endoscopy sooner. In these cases two pathologists will be asked to double check the biopsy changes and the hospital specialist will decide how soon the endoscopy test should be repeated.

Endoscopy is generally a safe procedure, but carries a small risk of complications. These occur when something goes wrong. Possible complications are bleeding or perforation (tear through the wall of the gullet or stomach), but they are rare occurring in less than 1 every 1000 endoscopies. It is important to understand this risk when agreeing to receive regular endoscopic monitoring. Also, endoscopy can be unpleasant, but an injection prior to the test (sedation) can make it much more tolerable.

What treatment is available for early cancer?
If severe cellular changes (high grade dysplasia) or a small cancer are found at endoscopy, a treatment may then be offered. Whenever possible, rather than an operation patients are offered endoscopic treatment as this is less invasive. Endoscopic treatment is performed through the channels present in the flexible camera tube in order to remove the cancer (endoscopic resection) or ablate (burn off) the Barrett's oesophagus using a treatment such as radiofrequency ablation. In some circumstances surgery may be needed when the cancer is more advanced.

What medication should I take?
Patients with Barrett's oesophagus are usually prescribed medications to control the acid reflux. The most common type of medication prescribed is called a proton pump inhibitor or more simply PPI. PPI is a safe drug and can be taken for many years without significant risks. Patients that are intolerant to PPI can be offered keyhole surgery to correct the hiatus hernia and stop the reflux to occur. Studies have showed that PPI and keyhole surgery are equally effective in controlling the reflux. However there is lack of evidence that PPI or keyhole surgery can prevent cancer from

occurring. Other medications include H2 blockers and drugs to neutralise the acid such as gaviscon or rennies. Sometimes more than one type of medication is recommended for use at the same time.

Does it matter what I eat?
There are no precise dietary recommendations for patients with Barrett's oesophagus. However, you should avoid foods if they make your reflux or heartburn symptoms worse. For example, excess of alcohol, coffee, chocolate and citrus fruits all fall into this category. Fatty foods also tend to take longer to leave the stomach and this can make patients feel uncomfortable. If you find that large meals irritate your Barrett's, then eating smaller amounts more often might suit you better. Overall, eat foods that suit you and enjoy all things in moderation!

If you need more information ask your family doctor or hospital specialist or visit this website http://www.h-cas.org/