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Barrett's Oesophagus Clinical Guidelines

February 2015

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Document Detail	
Document type	Clinical Guideline
Document name	Barrett's Oesophagus Clinical Guidelines
Version	1.0
Effective from	January 2015
Review date	June 2016
Owner	LCA
Author(s)	Dr Jason Dunn (Jason.Dunn@gstt.nhs.uk), Dr Jonathan Hoare, Dr Sameer Zar
Approved by, date	
Superseded documents	
Related documents	
Keywords	Barrett's oesophagus, dysplasia, acetic acid, surveillance, aneuploidy, P53
Relevant external law, regulation, standards	

Change History		
Date	Change details, since approval	Approved by

Contents

1 Summary

Oesophageal cancer is the fifth commonest cause of cancer death in the UK, and the incidence of adenocarcinoma (OAC) is rising. Patients are often without symptoms until the tumour has grown to be inoperable, and the survival for this cancer remains poor. Early diagnosis is crucial to improve survival.

Barrett's oesophagus (BE) is the pre-cancerous lesion. Recent British Society of Gastroenterology (BSG) guidelines recommend surveillance every 2–5 years depending on length of Barrett's segment. Endoscopic therapy is reserved for high risk Barrett's demonstrating high grade dysplasia (HGD), which carries a rate of progression to OAC of up to 60% over 5 years. The risk of progression of non-dysplastic Barrett's is low and unpredictable using standard histopathology techniques.

These guidelines have been compiled by the London Cancer Alliance (LCA) oesophago-gastric pathway group from BSG national guidelines and literature review.

2 Introduction

2.1 Background

Oesophageal cancer is the fifth commonest cause of cancer death in the UK, and the incidence of adenocarcinoma (OAC) is rising.[1] Patients are often without symptoms until the tumour has grown to be inoperable, and the survival for this cancer remains poor. Early diagnosis is crucial to improve survival.

Barrett's oesophagus (BE) is the pre-cancerous lesion. Recent BSG guidelines recommend surveillance every 2–5 years depending on length of Barrett's segment.[1] Endoscopic therapy is reserved for high risk Barrett's demonstrating high grade dysplasia (HGD), which carries a rate of progression to OAC of up to 60% over 5 years.

Techniques to treat HGD or intramucosal cancer include endoscopic mucosal resection (EMR), radiofrequency ablation (RFA) and, for small islands, argon plasma coagulation (APC). Photodynamic therapy has been superseded by RFA and is seldom used. These techniques should be performed at centres where endoscopic and surgical options can be offered to patients. All patients with dysplasia or early cancer, for whom therapy is considered, should be discussed at the specialist MDT for oesophago-gastric cancer. This team should include an interventional endoscopist, upper GI cancer surgeon, radiologist and a GI pathologist (minimum standard).

The guidance for low grade dysplasia (LGD) set out in this document is a departure from the BSG guidelines, with consideration for RFA at specialist centres in those with high risk features (genetic abnormalities). The organisation of surveillance lists across the LCA and mutual audit is discussed. Finally stratification for higher risk non-dysplastic BE is addressed, with a proposed new clinical trial of surveillance enhanced with other diagnostics (Barrett's surveillance EDGE).

2.2 Definition

Diagnosis of Barrett's oesophagus is described in the recent BSG guidance as:

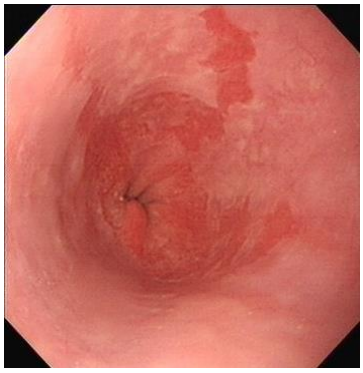
> *"an oesophagus in which any portion of the normal distal squamous epithelial lining has been replaced by metaplastic columnar epithelium, which is clearly visible endoscopically (≥1cm) above the GOJ and confirmed histopathologically from oesophageal biopsies (Recommendation grade C)."*

or

- presence of visible columnar mucosa above the top of gastric folds
- presence of intestinal metaplasia if length <3cm.

It is therefore essential to accurately document the level of the GOJ, which is often difficult as the majority of patients will have a hiatus hernia.[2] For the purposes of this guidance we recommend that the top of gastric folds (rather than extent of palisade vessels as is used in Japan) is the Z-line, and a measurement of columnar lined mucosa 1cm above this is sufficient for a diagnosis of Barrett's oesophagus (see Figure 1).

Figure 1 Barrett's oesophagus C0M3



On histopathological correlation there was evidence of specialised intestinal metaplasia, no dysplasia

(Courtesy of Dr Jason Dunn, GSTT)

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3 Surveillance Technique

3.1 Endoscopy

The BSG recommends that high-resolution endoscopy should be used in Barrett's oesophagus surveillance. Advanced imaging modalities, such as chromoendoscopy or 'virtual chromoendoscopy' (i.e. narrow band imaging (Olympus), I-scan (Pentax) or FICE (Fuji)) are not superior to standard white light endoscopy in Barrett's oesophagus surveillance and are therefore not recommended for routine use.[1]

Situations when these advanced techniques may be more advantageous are in patients where the intention is a target biopsy in the first instance. Such situations may include previous LGD/IND or those with very long segment Barrett's (i.e ≥10cm).

The technique for acetic acid chromoendoscopy is as follows:

1. Make up 20-50mls of 2.5% acetic acid (comes as 5% acetic acid from pharmacy, dilute 50:50 with water).
2. Wash entire mucosa with acetic acid using spray catheter.
3. Wash over surface with water.
4. Assess for areas of LAWS (loss of aceto-whitening sign).

Figure 2 Area of LAWS

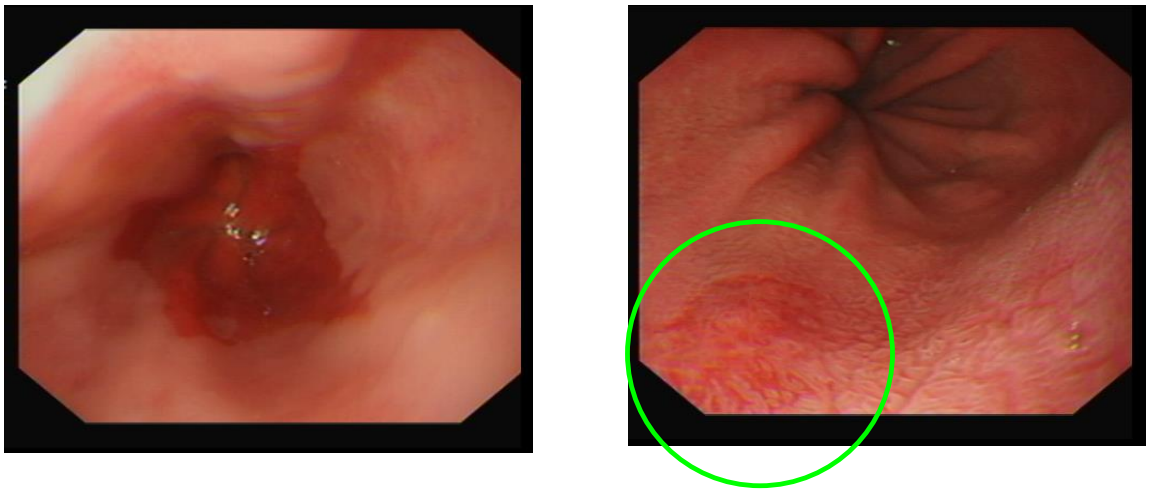


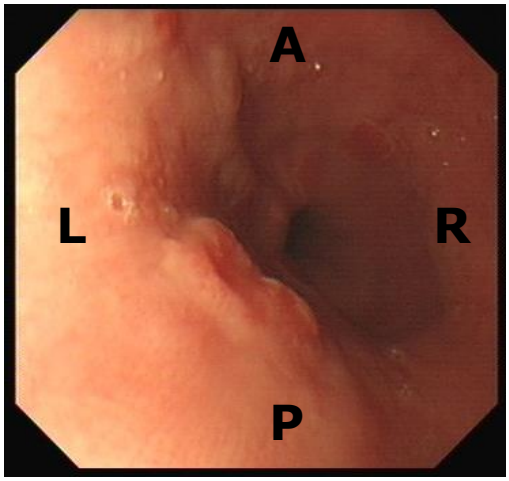
Image on left before acetic acid chromoendoscopy. On right shows area of loss of aceto-whitening within 30 seconds of application of acetic acid.

(Courtesy of Dr Jason Dunn, GSTT)

3.2 Lesion recognition

Target biopsy is recommended prior to embarking on the Seattle protocol four quadrant biopsy technique. This is primarily to target nodular disease (see Figure 3), with the proviso from the BSG guidance that 'visible lesions should be considered malignant until proven otherwise'. Nodules should be described according to the Paris classification (see Figure 4).

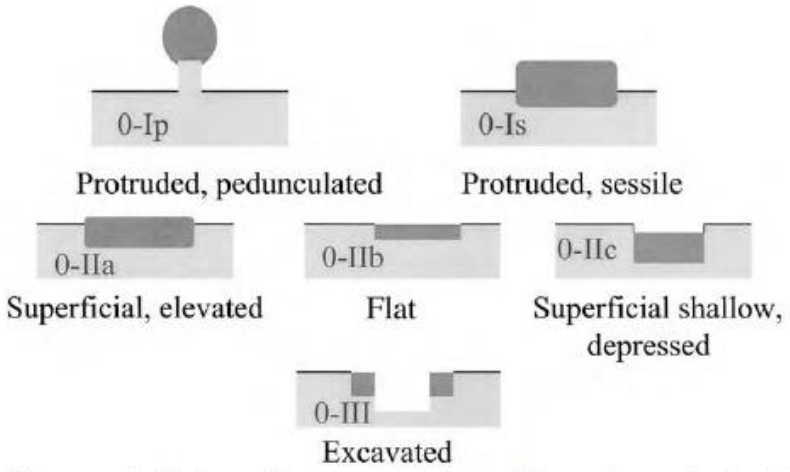
Figure 3 Nodule arising in Barrett's post treatment with RFA.



The lesion is at 32cm posterior/left wall or at 7 o'clock, Paris IIa/c

(Courtesy of Dr Jason Dunn, GSTT)

Figure 4 Paris Classification



Schematic representation of the major variants of *type 0* neoplastic lesions of the digestive tract: polypoid (Ip and Is), non-polypoid (IIa, IIb and IIc), non-polypoid and excavated (III). Terminology as proposed in a consensus macroscopic description of superficial neoplastic lesions.

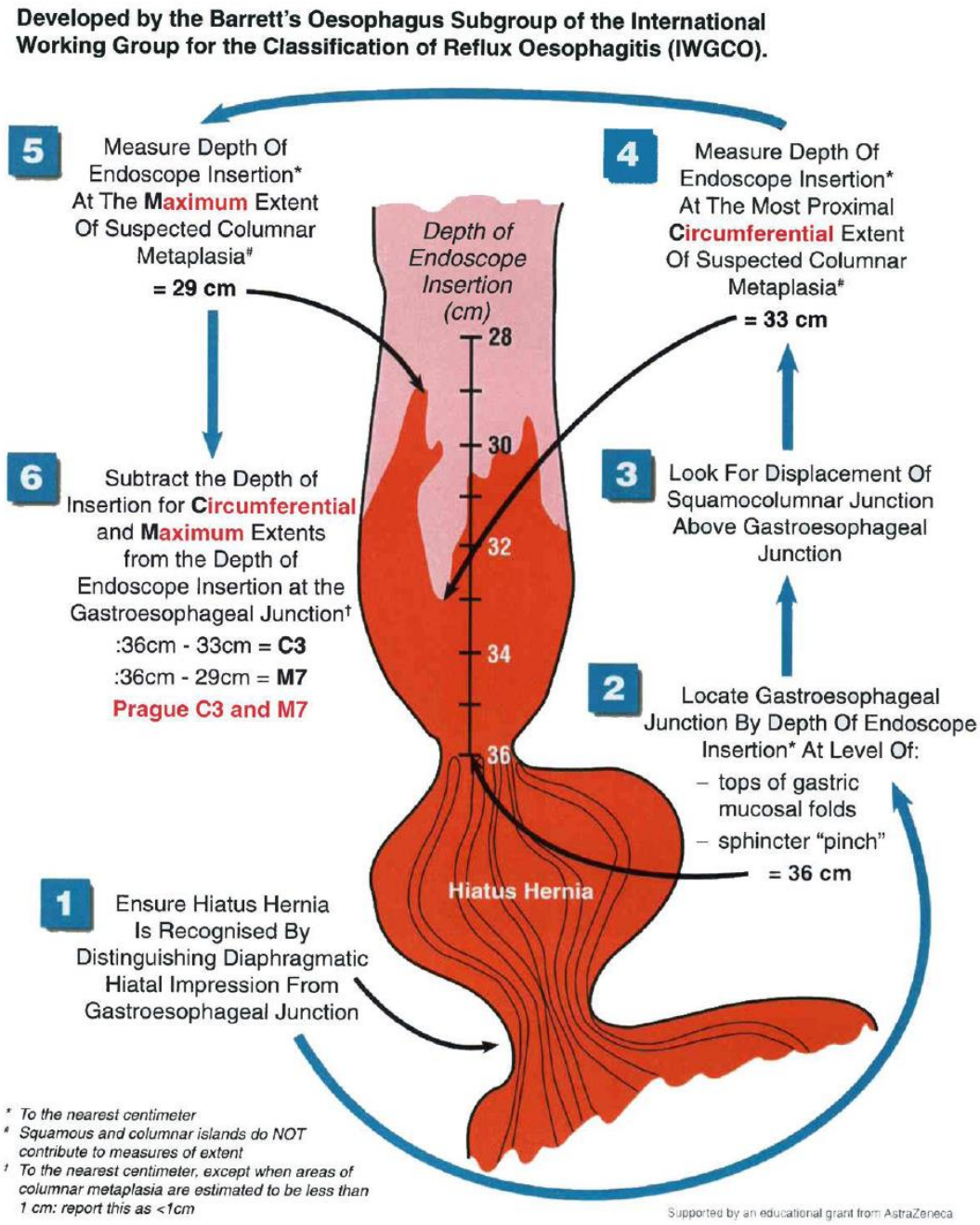
(Reproduced from The Paris endoscopic classification of superficial neoplastic lesions: esophagus, stomach, and colon. GIE 2003; 58(6) (SUPPL))

The presence of ulcers and strictures are also indicative of prevalent malignancy and should be reassessed without delay, including multiple targeted biopsies or diagnostic EMR if appropriate. If severe ulceration and stricturing is evident, but initial biopsy is negative for dysplasia, it is recommended to start high dose PPI (Nexium 40mg bd or equivalent) and reassess within 8 weeks.

To further accurately document the nature of Barrett's CLO the International Working Group for Classification of Oesophagitis (IWGCO) developed the Prague C&M criteria.[2] This scoring system is based on the circumferential (C value, in cm) and the maximal extent (M value, in cm) of BE above the gastro-

oesophageal junction (GOJ). For example, if BE was circumferential for 2cm above the GOJ and the maximal extent of non-circumferential BE was 5cm (i.e., with 3cm of BE "tongues") above the GOJ, this would be recorded as C2M5. The consensus group in this study decided that 'true islands of squamous and columnar mucosa should not influence the measurement of extent of BE and that only segments of contiguous BE are measured'. The proposed scoring system was validated in a study using 29 digital recordings of endoscopies. Internal validation yielded a high reliability coefficient value for agreement on the presence of BE >1cm (r =0.72).

Figure 5 Prague criteria – for endoscopically suspected oesophageal columnar metaplasia/Barrett's oesophagus



(Reproduced from – The Development and Validation of an Endoscopic Grading System for Barrett's Esophagus: The Prague C & M Criteria. Gastroenterology 2006;131(5):1392-9.)

3.3 Biopsy protocol

A four quadrant biopsy protocol (the 'Seattle' protocol) consisting of jumbo forceps biopsies from every 2cm of columnar mucosa, was first proposed for surveillance of patients with BE in 1993[3] and national guidelines of many countries recommend this protocol as standard.

Biopsies taken in 4 quadrants every 2cm through Barrett's segment. Individually labelled pots per level.

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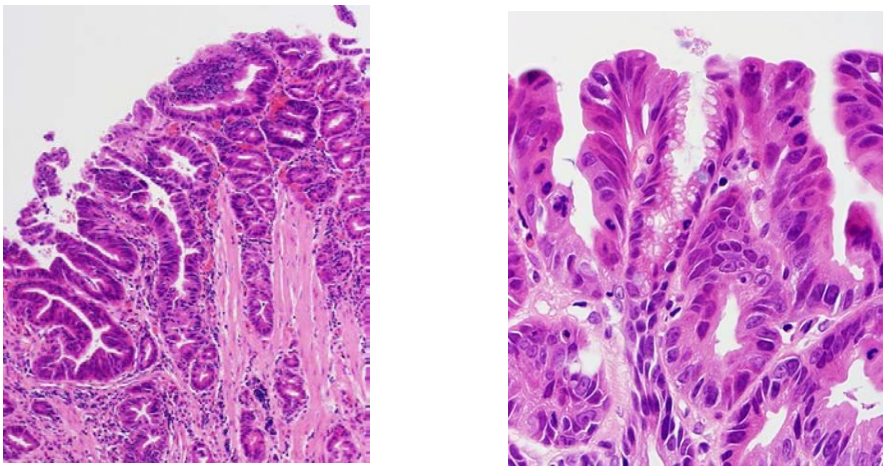
4 Management of Dysplasia

4.1 Management of high grade dysplasia

High grade dysplasia (HGD) is characterised by architectural changes which include increased budding, branching, and crowding, villiform surface configuration, and the presence of intraluminal bridges or papillae. Cytological features of HGD include marked nuclear pleomorphism (i.e., variation in nuclear size and shape), loss of polarity (i.e., loss of normal nuclear orientation, in which the long axis of the nucleus is perpendicular to the basement membrane and basally oriented), and full-thickness nuclear stratification (see Figure 6). Mitotic figures, especially atypical ones, are often present and may involve the surface epithelium. The diagnosis is subject to low inter-observer variability between pathologists, with studies demonstrating agreement 80% of the time.[1]

Figure 6 Haemotoxylin and eosin stain of high grade dysplasia (left x100, right x 400)



(Courtesy of Professor Marco Novelli, UCLH)

HGD is at present the most robust routinely used clinical marker of cancer progression in Barrett's oesophagus; its presence confers a 16-59% risk of developing cancer within 5 years of the diagnosis of

HGD.[2-5] it is generally accepted that a diagnosis of HGD is an indication for treatment and the BSG guidance, from 2005, states the following:

> *"High-grade dysplasia is associated with a focus of invasive adenocarcinoma in 30–40% of patients. For this reason,* ***if the changes persist after intensive acid suppression and are confirmed by two expert pathologists,*** *oesophagectomy in a specialised unit is currently recommended in patients considered fit for surgery.*
>
> *"(Recommendation grade C)."* [6]

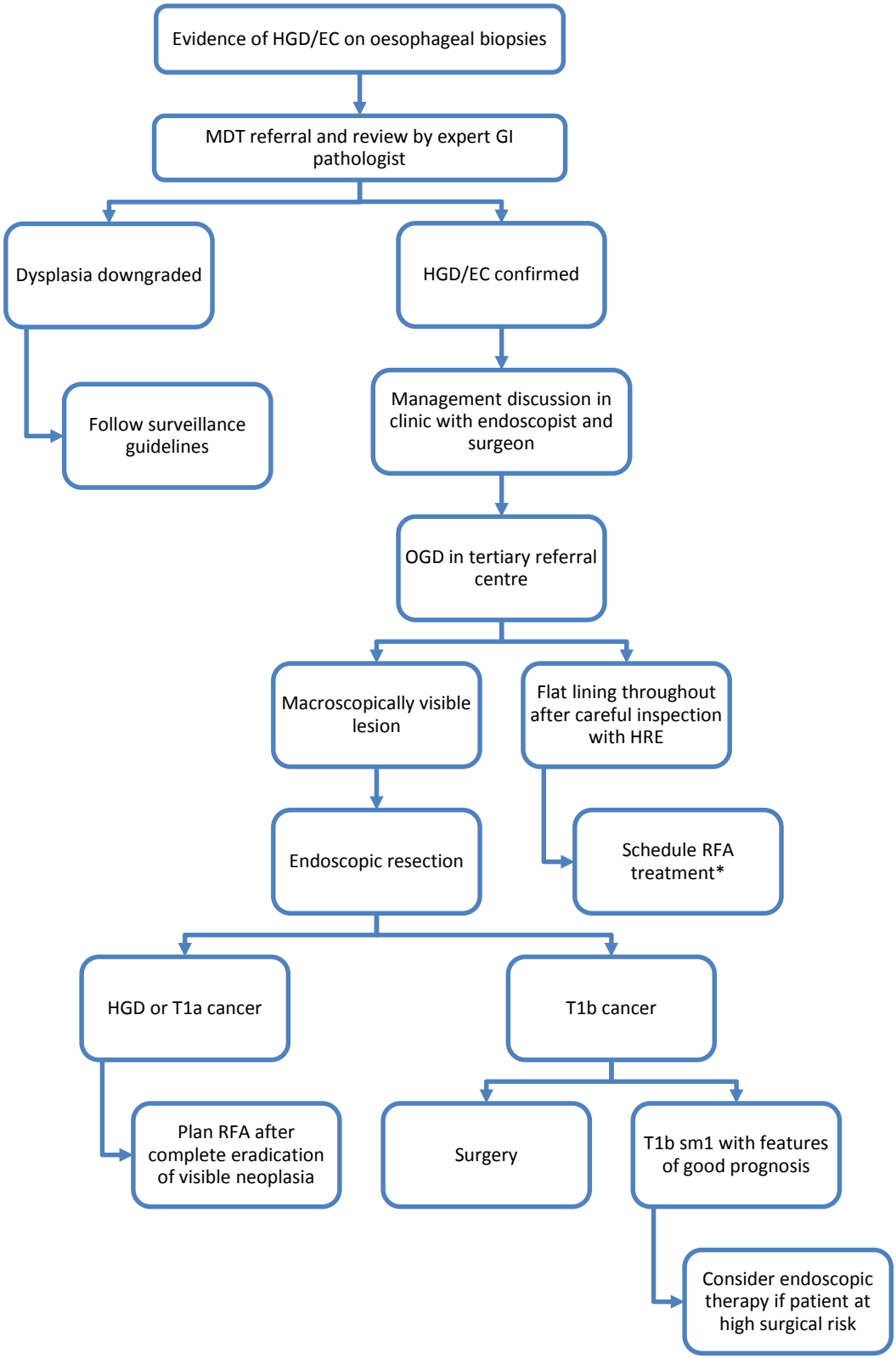
Two important changes were made in the recently updated 2013 version (Figure 7). One is the timing of referral to tertiary referral centre. These guidelines state that **a diagnosis of HGD on one occasion is sufficient for referral to UGI MDT for evaluation**. The reason is that these patients will often have more severe disease. Nodules or changes in surface pit pattern are often apparent in HGD, and can be removed by endoscopic mucosal resection (EMR) which will result in upstaging to cancer 40% of the time. High resolution endoscopy undertaken by specialist endoscopists may improve the pick up rate of visible lesions. An eight week period of intensive acid suppression therefore delays accurate staging, and subsequent potential for minimally invasive therapy (EMR/RFA).

The second important change is the adoption of minimally invasive therapy as a first line treatment for HGD or IMC arising in Barrett's oesophagus. This makes accurate early staging by an endoscopist with a special interest in RFA/EMR a critical step in the patient pathway.

Within the LCA, it is recommended that all patients with a diagnosis of HGD arising in Barrett's oesophagus **on one occasion** are referred to one of the three centres that currently undertake RFA/EMR (Guy's & St Thomas' NHS Foundation Trust, Imperial College Healthcare NHS Trust or The Royal Marsden NHS Foundation Trust). The National Oesophago-Gastric Cancer Audit now includes HGD, and forms are shown in Appendix A.

A patient with HGD should be referred at first diagnosis to a centre MDT.

Figure 7 BSG guidelines for management of HGD 2013

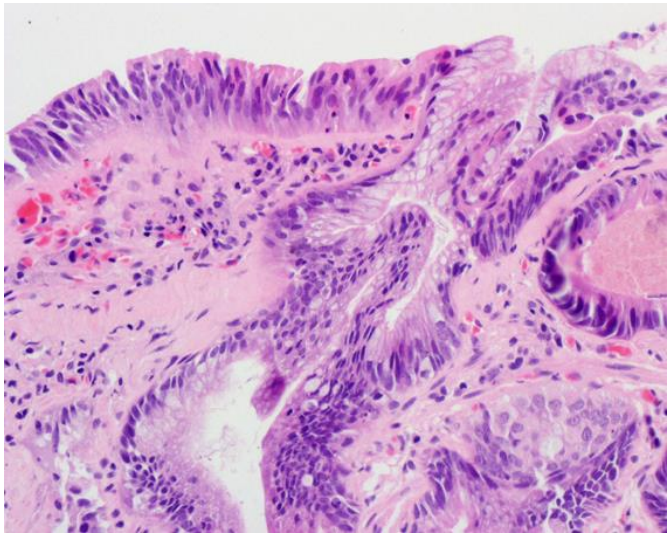


*** Repeat mapping biopsies may be useful to understand the spatial extent of the dysplasia; however repeat evidence of HGD is not necessary to initiate treatment pathway due to sampling error.**

4.2 Management of low grade dysplasia/indefinite for dysplasia

Low grade dysplasia (LGD) is characterised by crypts with relative preservation of simple glandular architecture. Epithelial cell nuclei are oval or elongated and generally retain polarity. The nuclei are hyperchromatic with mild irregularity of nuclear membrane contour. Nuclear stratification is present and usually occupies the lower half of the thickness of the epithelium; full-thickness stratification is not present (see Figure 8). Other features include mucin depletion, decreased number of goblet cells, and increased epithelial mitotic figures. Importantly, there is lack of maturation at the surface such that these changes are present on surface epithelium.

Figure 8 Haemotoxylin and eosin stain of low grade dysplasia



(Courtesy of Professor Marco Novelli, UCLH)

Less is known about the natural history of LGD, with risk of progression to cancer varying between 0.6-13.4% per patient year in surveillance cohorts (see Table 1).[7-14] This compares to 0.3-0.6% for non-dysplastic BE (NDBE).[15] One of the explanations could be the high degree of interobserver variability in establishing this diagnosis between pathologists.[16] Skacel *et al*. reported that when two or more histopathologists agree on a diagnosis of LGD, the risk of HGD/OAC progression increases.[17] Seven out of 43 patients developed neoplasia with a single pathologist reporting LGD; if two agreed the risk rose to 41%[18,19] and if all three agreed there was an 80% risk.[20,21] These conclusions were drawn from a relatively small number of patients with LGD.

Table 1 Risk of developing cancer/high grade dysplasia per patient year with low grade dysplasia

Study	Number patients	Risk of cancer	Risk of HGD	Comments
Reid	43	2.4%		Increased risk with aneuploidy
Weston	54	3%		
Skacel	43	3.7%	12.9%	
Sharma	156	0.6%		
Lim	34	3.4%		
Gatenby	217	2.7%	4.6%	Reduced to 1.4% and 2.2% when prevalent cases excluded
Wani	611	1.7%		(meta-analysis)

Curvers (downstaged)	92	0.49%	Unclear how many were prevalent HGD/OAC
Curvers (consensus)	19	13.4%	

A more recent study by Curvers *et al* confirms the importance of consensus diagnosis for the management of LGD.[11] Biopsies from 147 patients with LGD diagnosed at a local hospital were reviewed by two expert GI pathologists. After review 85% of the patients were downstaged to non-dysplastic BE (NDBE) or indefinite for dysplasia. In only 15% of the patients was the initial diagnosis confirmed as LGD. After a mean 51 months follow up, patients with a consensus diagnosis of LGD had a higher incidence rate of HGD or OAC than those downstaged to NDBE (13.4% vs 0.49% per patient per year). These findings need validation in larger prospective studies.

The uncertainty of the risk of cancer has led to great debate about the advantages and disadvantages of treating low grade dysplasia, particularly with the advent of minimally invasive endoscopic therapies. There is an argument that ablation of LGD is more cost effective than surveillance, with models demonstrating a 65% reduction in progression if complete reversal of dysplasia (CR-D) is achieved in 28%.[22] A feature of LGD that is not included in these models is spontaneous regression. This is demonstrated in the recent RCT of RFA versus a sham procedure, when 23% of patients achieved spontaneous regression of low grade dysplasia in the control group.[23] This is most likely related to sampling error, the inter-observer variability among pathologists, misdiagnosis, removal of the dysplastic focus by biopsy, and perhaps even true regression of the dysplastic area. In 2005 the BSG recommended that a diagnosis of LGD should warrant close follow-up, with endoscopy every 6 months. This approach has been shown to increase detection of HGD/OAC in a registry setting.[24]

The results of a multicentre RCT for RFA compared with endoscopic surveillance in a large cohort of patients with LGD have recently been published. Over 500 patients with LGD were assessed for entry into this study, but of these only 136 were eligible for entry with confirmed LGD on two occasions by two pathologists. The results demonstrated 27% of the surveillance population went on to develop HGD/cancer vs 1% in the RFA group. The trial was stopped early because of this.

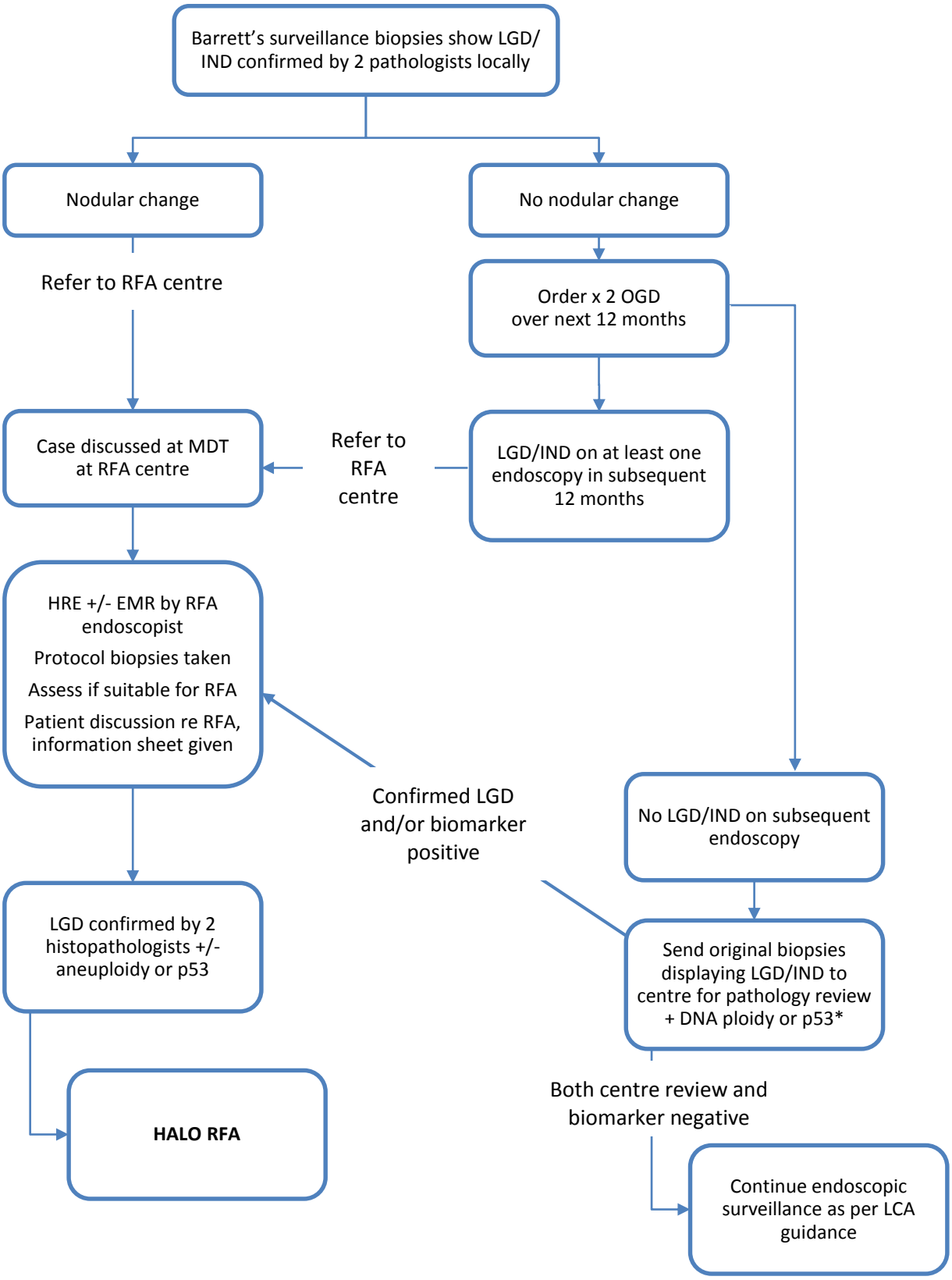
Due to this study the BSG guidance changed in 2014 with an addendum published recently on the management of LGD. It recommends that if LGD is confirmed by two pathologists on two separate occasions then patient should be considered for endoscopic therapy.

A patient with LGD/IND should have two further endoscopies over the next 12 months, 6 months apart.

If LGD/IND is confirmed a second time in the subsequent 12 months after initial diagnosis, then refer to centre for consideration of EMR/RFA.

The use of DNA ploidy (aneuploidy) by image cytometry (ICM), which has been shown to significantly increase the odds ratio of developing HGD/OAC in a LGD cohort[25], is advocated for use across the LCA. ICM has been validated against the gold standard of flow cytometry, but is advantageous as it can be undertaken on formalin fixed paraffin embedded (FFPE) tissue, and hence routine biopsy samples that have been archived.[26] ICM is available at two pathology labs in London, one in the London Cancer Alliance region (Guy's and St Thomas' NHS Foundation Trust, Professor Eddy Odell) and one in the London Cancer area (UCLH, Professor Marco Novelli). A flowchart for management of LGD is shown in Figure 9.

Figure 9 Flowchart for management of LGD



*** dependent on availability**

(Courtesy of Dr Jason Dunn, GSTT)

4.3 Management of non-dysplastic Barrett's

This group of patients makes up the majority undergoing surveillance. Recent BSG guidance has modified the interval for follow up and is shown below. An important change to the 2005 guidance is the surveillance interval, which is based on length. If the Barrett's segment is <3cm then a 3-5 year follow up is recommended, and if ≥3cm then a 2-3 year follow up. The variable length of follow up is somewhat contentious, and the LCA guidance uses the longer length of follow up, i.e. 3 year follow up for longer segments and 5 year for short, with the proviso that surveillance will be in accordance to the above biopsy protocol. If the patient has a very long segment (≥ 10cm) consideration should be made to refer to a local centre where acetic acid and high resolution endoscopy (HRE) is available for ongoing surveillance. Full BSG guidance is available at the following link:
www.bsg.org.uk/clinical-guidelines/oesophageal/guidelines-on-the-diagnosis-and-management-of-barrett-s-oesophagus.html

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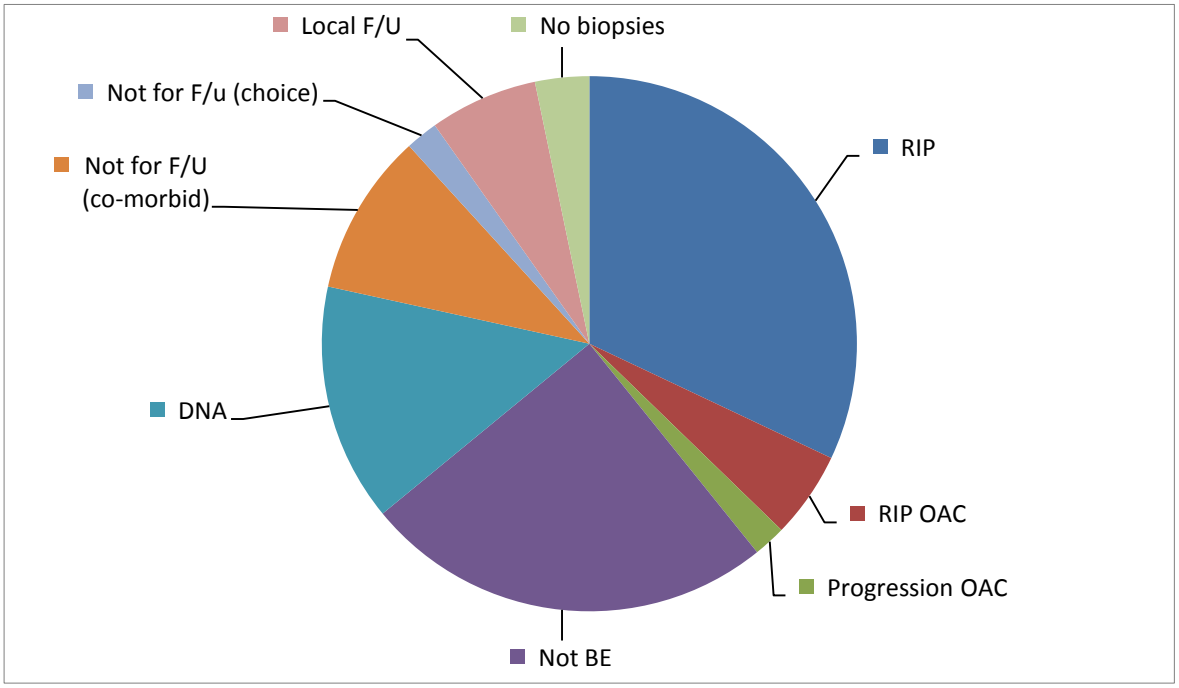
5 Surveillance Programme Exit

When surgery was the recommended first line treatment for HGD, it was recommended that if the patient was not fit for surgery they should not continue in a surveillance programme. With the introduction of minimally invasive endoscopic therapy, age is now less of a factor for treatment and therefore ongoing surveillance, with the mean age from the recent UK RFA registry 68.1 +/- 9.6 years, and the maximum age 91 years old. It also should be noted that endoscopic therapy is a long process with multiple endoscopies over that time. The median is 2.5 treatments, and these at 3 month intervals, so including initial staging endoscopy this equates to four endoscopies over a year. In addition, the follow up is 6 monthly over the following 2 years, then annual indefinitely thereafter.

The following diagram (Figure 10) shows 155/579 patients who exited from the GSTT Barrett's surveillance programme over a 5-year period (unpublished data). The majority either die from other causes or do not have Barrett's oesophagus (short segments with no IM). Only 5% died from oesophageal cancer.

Figure 10 Barrett's surveillance programme exit at GSTT



(Courtesy of Dr Jason Dunn, GSTT)

6 Environment

Organisation of surveillance programmes is key to structured follow up. Non-adherence has been reported as high as 50% in a US community setting and is associated with significantly decreased dysplasia detection. There is a growing body of evidence that dedicated surveillance lists improve endoscopic technique and dysplasia detection rate (DDR). In the recent AspECT study there was a significant increase in four quadrant biopsy technique (32% to 86%) and number of biopsies taken post trial adoption at site.[1] A recent report from the UKBOR cohort on 817 patients demonstrated a large proportion of dysplastic disease (>90%) was detected on specific surveillance endoscopies, though variation in surveillance practice for BE was observed throughout the UK.[2] A study by Abela *et al.* in a UK setting showed that there was a 13-fold increase in detection of prevalent dysplasia between patients who underwent four quadrant biopsies every 2cm

(median biopsy number 16) compared with those who had non-systematic biopsies (median biopsy number 4).[3] This study showed an increased DDR: 18.9% vs 1.6% for LGD; 2.8% vs 0% for HGD.

It is suggested that each of the 15 LCA provider organisations has a dedicated list for Barrett's surveillance, with two slots for long segments (>3cm) and one slot for short segments. The frequency will be dependent on the size of population currently in surveillance at each site, although it is predicted from introduction of dedicated lists in hospitals in the region to be no more than 1–2 lists per month.

Finally a nominated lead for Barrett's surveillance per provider is recommended to manage the programme. It would be envisaged that this would be the same as the upper GI cancer lead at each centre.

References

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7 Audit and Research

7.1 Audit

One role of the lead at each centre would be the organisation of an annual audit which will be rolled out throughout the LCA. Auditable criteria include the following:

- Prague documentation
  - Aim for >80%
- Number of bx per 2cm
  - Aim for >70%
- DDR
  - Aim at least 7% for all grades
- Dual pathology review of LGD/IND
  - 90%
- Central pathology review of high grade dysplasia
  - 100%
- Time to centre referral for HGD (one only x 2 patholologists review) or persistent IND/LGD (2 times, x 2 pathologists review)
  - Within 2 weeks 90%
- Surveillance intervals correct according to guidelines
  - As above, 80%

7.2 Future LCA research projects – management of indeterminate risk groups

It is becoming evident from epidemiological studies that not all non-dysplastic Barrett's has the same risk of progression. Clinical factors that have been associated with an increased relative risk over baseline include length of Barrett's oesophagus, age, male sex, obesity, smoking and nocturnal reflux. Genetic factors have also been shown as biomarkers of risk, including DNA ploidy abnormalities and p53. It has been shown that patients without HGD who have changes in the total number of chromosomes in the Barrett's cells on flow cytometry (aneuploidy) have a 28% risk of developing cancer in the next five years.[1]

New studies are needed to move away from pathology alone as a risk stratification tool. One barrier is the large cohort of patients that will not progress to cancer, and therefore biomarker studies have been limited to phase 3 retrospective case control studies.

A new study evaluating enhanced surveillance – Barrett's surveillance EDGE (**E**nhanced according to **D**emographic, **G**enetic and **E**ndoscopic appearances) is taking place at Guy's and St Thomas' NHS Foundation Trust, with a view to roll out across the LCA. The aim of this cross-sectional study is to evaluate genetic and endoscopic markers of risk of progression, in those at greater risk of progression using clinical parameters. The following are suggested based on epidemiological data.

1) LGD or IND confirmed by 2 pathologists on one occasion
2) NDBE and family Hx of OAC (1st degree relative)
3) Residual or neo-BE post oesophagectomy for OAC
4) BE > 6cm maximal extent

Genetic linkage studies have demonstrated a 2-fold increase risk of cancer in siblings[2], though no biomarker studies have been done on these patients. Finally a recent paper demonstrated that as the length of non-dysplastic Barrett's increases, so does the risk of progression.[3] When >6cm the OR = 5.96 (CI 1.9-16.7), and is almost double again when >13cm OR = 10.27 (CI 1.9-53.7.7).

When enrolled, patients would undergo annual follow up at a centre for a total of 3 years, with acetic acid, high resolution endoscopy, multiple biopsy and biomarkers. The expected frequency of the clinical risk parameters occurring has been assessed in the GSTT cohort of Barrett's surveillance (unpublished data). Over a 2 year time period (Oct 2011-13), 376 patients were seen. 18/376 had IND/LGD, 3/376 had a positive family history, 5/376 residual or neo-BE post surgery for OAC and 36/376 had NDBE >6cm. Total number of patients = 51, or 13.5% of surveillance population.

References

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8 Conclusion

The above guidance has been developed to improve the treatment of patients with Barrett's oesophagus throughout the LCA. By introducing these guidelines the overarching aim is to improve the detection rate of dysplasia and early cancer, in order to treat with endoscopic therapy when the disease is potentially curable. By rationalising surveillance lists, redefining referral pathways for dysplasia and the introduction of a new pilot study to target higher risk cohorts of patients, it would be hoped that this would create a highly efficient system to allow equity of treatment for all patients in LCA centres. Finally both audit and research data generated from this work should contribute to the evidence base for surveillance and treatment of Barrett's oesophagus nationally.

Appendix A: NOGC Audit – Patients with HGD

National Oesophago-Gastric Cancer Audit
New Patient Registration sheet
Patients with Oesophageal High Grade Glandular Dysplasia

Patient Details

Surname: _____ Forename: _____
NHS number: _____ Postcode: _____
Sex: Male ☐ Female ☐ Not specified ☐ Date of birth: _____

Initial Referral to Local Oesophago-gastric Team and Diagnostic Process

Source of referral
From surveillance service: ☐ Symptomatic referral ☐ Not known ☐
Date of endoscopic biopsy in which HGD was first diagnosed: _____
Hospital where the endoscopic biopsy was taken: _____
Was a second biopsy performed? Yes ☐ No ☐
Did the second biopsy show HGD? Yes ☐ No ☐

Endoscopic Report

HGD appearance
Flat mucosa ☐ Nodular lesion ☐ Depressed lesion ☐ Not known ☐
Barrett's Segment
Present ☐ Absent ☐ Not known ☐
Length of Barrett's Segment, if present
Length of ***Circumferential*** Columnar Lining (nearest 0.5 cm): C ____.___cm
Maximum length including tongues/islands of Columnar Lining (nearest 0.5 cm): M ____.___cm
HGD Lesion (based on pathology report)
Unifocal ☐ Multi-focal ☐ Not known ☐
Was diagnosis confirmed by second pathologist? Yes ☐ No ☐ Not known ☐

Planned Treatment

Hospital at which treatment plan made _____
Date treatment plan agreed _____
Was the treatment plan agreed at an MDT meeting? Yes ☐ No ☐
Will the patient be referred to a specialist hospital for treatment? Yes ☐ No ☐ Not applicable ☐

1

Planned treatment modality			
Surveillance	☐	Radiofrequency ablation	☐
Oesophagectomy	☐	Argon plasma coagulation	☐
Photo dynamic therapy	☐	Multipolar electrocautery	☐
Endoscopic Mucosal Resection (EMR)	☐	Laser therapy	☐
Endoscopic Submucosal Dissection (ESD)	☐	Cryotherapy	☐
Other treatment	☐	No treatment	☐

Use of Endoscopic Mucosal Resection (EMR) / Endoscopic Submucosal Dissection (ESD)			
EMR/ESD was not performed:	☐	Performed for diagnostic purpose:	☐
Performed for therapeutic purpose:	☐	Performed for both diagnostic and therapeutic purpose:	☐

Date of EMR/ESD: _____

Results of EMR/ESD:			
Complete excision:	☐	Incomplete, follow up Oesophagectomy	☐
Incomplete, follow up surveillance	☐	Incomplete, follow up EMR/ESD	☐
		Complete excision, follow up with endoscopic therapy	☐

Post-treatment Histology (pathology results based on EMR/ESD)	
No high-grade dysplasia or carcinoma	☐
High-grade dysplasia confirmed	☐
Intramucosal carcinoma identified	☐
Submucosal carcinoma or worse	☐

Published by London Cancer Alliance

London Cancer Alliance

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London SW1H 0QS

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