AUGIS Summer Newsletter 2019

Full Report from AUGIS Lead for Non-Malignant OG Diseases

Professor Ashraf Rasheed

Following on from the winter newsletter i.e. making it informative, I will try to provide a summary on a selected OG matter and in this newsletter, I chose Barrett's Oesophagus, specifically in relation to the available clinical standards for radiofrequency ablation (RFA), endoscopic mucosal resection (EMR) and submucosal dissection (ESD). This will be my last newsletter to touch on pre-malignant or malignant OG matters as my colleague James Gossage will be the dedicated lead for the OG Cancer and I will limit myself to non-malignant OG diseases. I will list the full recommendations from each guidance/guideline in a chronological order to show case the evolutionary process of our understanding and its translation into recommendations which and I believe it to be an essential knowledge to every UGI clinician. I will then make few final comments of my own.

Barrett's Epidemiology in the UK

To set the scene, it is useful to look at the UK epidemiology of BO; Barrett's oesophagus is prevalent in 1.5–2.5% of the adult UK population[1] with around 60,000 new cases per year (annual incidence around 0.1%). In around 60% of cases, Barrett's oesophagus seem to be associated with chronic gastro-oesophageal reflux[2] and is found in 15–20% of adults undergoing endoscopic investigation for symptomatic chronic reflux. The condition can develop in the absence of symptoms and only 5–10% of adults with reflux develop Barrett's oesophagus[3]. Other factors associated with increased risk of developing Barrett's oesophagus are Caucasian race, male sex, and older age.[1,3]

Men with Barrett's oesophagus have an absolute lifetime risk of developing oesophageal adeno-carcinoma of about 5% compared with 3% for women[3]. In studies of Barrett's oesophagus patients with flat HGD undergoing surveillance, approximately six patients per 100 patient-years develop oesophageal adenocarcinoma. The combined incidence of HGD and oesophageal adenocarcinoma in patients under surveillance is estimated to be higher in the UK (13.0/1,000 patient-years; 95% CI 7.4 to 22.8) than in other European countries (7.3/1,000 patient-years; 95% CI 3.6 to 23 15.0)[5].

The rate of progression to cancer among patients with Barrett's oesophagus in the UK as a whole is approximately 1% per year[1]. The average risk of mortality attributable to oesophageal adenocarcinoma among Barrett's oesophagus patients under surveillance has been estimated at 0.3% per year (incidence 3.0/1,000 patient-years; 95% CI 2.2 to 3.9)[4].

NICE Guidance

I. Epithelial Radiofrequency Ablation for Barrett's 0esophagus Interventional Procedures Guideline (IPG344], Published 26 May 2010

1. RFA may be used in patients with Barrett's oesophagus with low-grade dysplasia (LGD) with normal arrangements for clinical governance, consent and audit or research.

2. RFA should only be used in patients with no dysplasia in the context of research.

3. Patient selection for RFA ablation for Barrett's oesophagus with low-grade dysplasia should be done by a multidisciplinary team experienced in the management of Barrett's oesophagus, as described in the BSG guideline.

4. Endoscopic radiofrequency ablation for Barrett's oesophagus with low-grade dysplasia should only be done by endoscopists with specific training in this procedure, as described in the BSG guideline.

5. Clinicians should enter details of all patients undergoing endoscopic radiofrequency ablation for Barrett's oesophagus with low-grade dysplasia or no dysplasia onto the UK national HALO patient registry and review clinical outcomes locally.

6. NICE encourages further research into endoscopic radiofrequency ablation for Barrett's oesophagus with no dysplasia. Studies should define clearly the policies used for histological diagnosis. Outcomes should include complete resolution of Barrett's oesophagus, change and progression to low-grade dysplasia, high-grade dysplasia or cancer. All complications should be reported, particularly development of strictures. Comparative studies against surveillance would be useful

II. Endoscopic Submucosal Dissection of Oesophageal Dysplasia and Neoplasia, Interventional Procedures Guidance [IPG355] Published date: September 2010.

1. Current evidence on the efficacy of endoscopic submucosal dissection (ESD) in patients with oesophageal adenocarcinoma or high-grade dysplasia in Barrett's oesophagus is limited in quantity and there are safety concerns specifically regarding the risk of oesophageal perforation. Therefore, in these patients, the procedure should only be used in the context of research.

2. Current evidence on the efficacy of ESD in patients with oesophageal squamous carcinoma or squamous dysplasia is limited. This evidence is mostly from Japan where the epidemiology of oesophageal cancer is different from the UK. There are safety concerns specifically regarding the risk of oesophageal perforation. Therefore, in these patients, the procedure should only be used with special arrangements for clinical governance, consent and audit or research.

3. Clinicians wishing to undertake ESD for oesophageal squamous carcinoma or squamous dysplasia should take the following actions:

- Inform the clinical governance leads in their Trusts
- Ensure that patients and their carers understand the uncertainty about the procedure's safety and efficacy and provide them with clear written information. In addition, the use of NICE's information for patients ('Understanding NICE guidance') is recommended.
- Audit and review clinical outcomes of all patients having ESD for oesophageal squamous carcinoma or squamous dysplasia.
- Patient selection should be carried out by an upper gastrointestinal cancer multidisciplinary team.
- The procedure is technically challenging and should be carried out only by clinicians with specific training in the technique.
- NICE encourages further research into the procedure. Studies should define clearly the type, grade and stage of cancer or dysplasia being treated. Efficacy outcomes should include adequacy of resection and the proportion of patients free from local recurrence. Safety outcomes should include perforation and stricture, and the consequences of these complications

III. Barrett's Oesophagus: Ablative Therapy, Clinical Guideline [CG106] Published 11 August 2010 https://www.nice.org.uk/Guidance/CG106

Endoscopic Therapy

1. Before considering endoscopic therapy as an alternative to surgery, a confirmed diagnosis of high grade dysplasia or intra-mucosal cancer in Barrett's oesophagus should be agreed by a designated specialist multidisciplinary team for oesophago-gastric cancer.

2. All treatments for high-grade dysplasia and intra-mucosal cancer in Barrett's oesophagus should be performed by specialist oesophago-gastric cancer teams with the experience and facilities to deliver the treatments recommended in this guideline.

3. Offer endoscopic therapy as an alternative to oesophagectomy to people with high-grade dysplasia and intra-mucosal cancer (T1a), taking into account individual patient preferences and general health. Endoscopic therapy is particularly suitable for patients who are considered unsuitable for surgery or who do not wish to undergo oesophagectomy. Endoscopic mucosal resection.

4. Consider using endoscopic mucosal resection alone to treat localised lesions.

5. Use circumferential endoscopic mucosal resection with care because of the high incidence of stricture formation.

Endoscopic Ablative Therapy

1. If residual or recurrent disease is suspected, consider additional or repeated therapy with appropriate follow-up using: endoscopic mucosal resection with further pathological assessment or ablative therapy (radiofrequency ablation or photodynamic therapy) or endoscopic mucosal resection and ablative therapy (radiofrequency ablation, argon plasma coagulation or photodynamic therapy).

2. Consider using radiofrequency ablation alone or photodynamic therapy alone for flat high-grade dysplasia, taking into account the evidence of their long-term efficacy, cost and complication rates.

3. Do not use argon plasma coagulation, laser ablation or multipolar electrocoagulation alone, or in combination with each other, unless as part of a clinical trial.

Endoscopic Mucosal Resection in Combination with Ablative Therapies

If using endoscopic mucosal resection, consider following with an additional ablative therapy (radiofrequency ablation, argon plasma coagulation or photodynamic therapy) to completely remove residual flat dysplasia, taking into consideration the side-effect profiles.

IV. Endoscopic Radiofrequency Ablation for Barrett's Oesophagus with Low-Grade Dysplasia or No Dysplasia, Interventional Procedures Guideline (IPG496] Published 23 July 2014) https://www.nice.org.uk/guidance/ipg496

1. Barrett's oesophagus is a precancerous condition characterised by abnormal replacement of the squamous epithelium of the lower oesophagus by a type of columnar epithelium resembling that in the stomach and intestine.

2. In some patients, Barrett's oesophagus may progress through a series of stages to oesophageal adenocarcinoma – a cancer with a poor prognosis. These intermediate stages are graded into low-grade and high-grade dysplasia according to the degree of abnormal cellular architecture.

3. The risk of progression to oesophageal adenocarcinoma for any individual with Barrett's oesophagus is difficult to predict accurately. In general, the risk of cancer is highest for patients with high-grade dysplasia, lower for patients with low-grade dysplasia, and lowest for patients with no dysplasia (also referred to as intestinal metaplasia – a change from epithelium that is normal for this site but with no evidence of dysplasia). Accurate classification of Barrett's oesophagus into these distinct histopathological types is difficult; there is the possibility of diagnostic misclassification because of biopsy sampling error and subjective biopsy interpretation. Strategies for addressing this include multiple biopsy sampling, diagnosis on at least 2 occasions, confirmation by 2 specialist histopathological

experts and confirmation by an independent pathologist external to the original institution each time – all in the context of a multidisciplinary team.

4. The main risk factor for developing Barrett's oesophagus is a history of reflux of acid and bile into the oesophagus. Reflux commonly produces symptoms of heartburn but it can be asymptomatic.

5. The management of Barrett's oesophagus is determined by the type of dysplasia present. In Barrett's oesophagus with no dysplasia or low-grade dysplasia, periodic endoscopic surveillance and repeat biopsies may be considered, with the aim of early detection of progression to high-grade dysplasia or cancer. If high-grade dysplasia or early cancer (carcinoma in situ) is detected, then treatment is recommended. If the disease is superficial (confined to the mucosa), treatment can usually be done endoscopically.

6. Endoscopic treatments for Barrett's oesophagus aim to destroy the Barrett's epithelium, leaving a surface that is subsequently replaced with a normal squamous epithelium. If the disease is flat, then it is generally ablated using one of several possible modalities, such as photodynamic therapy, argon plasma coagulation, laser ablation, cryotherapy or multipolar electrocoagulation. If there are visible abnormalities, such as nodules or ulcers, then those areas are usually removed by endoscopic resection.

7. Current evidence on the efficacy of endoscopic radiofrequency ablation for Barrett's oesophagus with low-grade dysplasia is adequate provided that patients are followed up in the long term. There are no major safety concerns. Therefore, this procedure may be used in patients with Barrett's oesophagus with low-grade dysplasia with normal arrangements for clinical governance, consent and audit or research.

8. Current evidence on the efficacy and safety of endoscopic radiofrequency ablation for Barrett's oesophagus with no dysplasia is limited in quality and quantity. Therefore, this procedure should only be used in patients with no dysplasia in the context of research.

9. Patient selection for endoscopic radiofrequency ablation for Barrett's oesophagus with low-grade dysplasia should be done by a multidisciplinary team experienced in managing Barrett's oesophagus, as described in the British Society of Gastroenterology guidelines.

10. Endoscopic radiofrequency ablation for Barrett's oesophagus with low-grade dysplasia should only be done by endoscopists experienced in treating Barrett's oesophagus, as described in the British Society of Gastroenterology guidelines.

11. Clinicians should enter details of all patients undergoing endoscopic radiofrequency ablation for Barrett's oesophagus with low-grade dysplasia or no dysplasia onto the UK National HALO Patient Registry, and review clinical outcomes locally.

12. NICE encourages further research into endoscopic radiofrequency ablation for Barrett's oesophagus with no dysplasia. Studies should define clearly the policies used for histological diagnosis. Outcomes should include complete resolution of Barrett's oesophagus, change and progression to low-grade dysplasia, high-grade dysplasia or cancer. All complications

should be reported, particularly development of strictures. Comparative studies against surveillance would be useful.

Other Published Guidelines/Guidance

I. British Society of Gastroenterology/ Fitzgerald RC, di Pietro M, Ragunath K, et al.: Guidelines on the Diagnosis and Management of Barrett's Oesophagus Gut. 2014; 63(1): 7–42. https://www.bsg.org.uk/resource/bsg-guidelines-on-the-diagnosis-and-management-of-barrett-s-oesophagus.html

Management of Dysplasia and Early Cancer

1. Patients with a diagnosis of indefinite for dysplasia should be managed with optimisation of anti-reflux medication and repeat endoscopy in 6 months. If no definite dysplasia is found on subsequent biopsies, then the surveillance strategy should follow the recommendation for non-dysplastic Barrett's oesophagus (*Recommendation grade C*).

2. Management of low-grade dysplasia (LGD) is unclear in view of limited data about the natural history. It is essential that the diagnosis is confirmed by two pathologists, and patients should be surveyed endoscopically at 6 monthly intervals. Currently, ablation therapy cannot be recommended routinely until more data are available (*Recommendation grade C*).

3. Expert high-resolution endoscopy (HRE) should be carried out in all Barrett's patients with biopsy-detected HGD in order to detect visible abnormalities suitable for endoscopic resection (ER) (*Recommendation grade B*).

4. Visible lesions should be considered malignant until proven otherwise (*Recommendation grade C*).

5. Description of lesion morphology using the Paris classification gives an indication of the likelihood of invasive cancer and aids communication between clinicians. This should

therefore be used for all visible lesions but cannot at present be used to predict prognosis (*Recommendation grade C*).

6. 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) (*Recommendation grade C*).

7. Patients with dysplasia or early cancer should be informed of treatment options and have access to consultation with all specialists as required (*Recommendation grade C*).

Endoscopic Therapy for Barrett's-Related Neoplasia

1. For HGD and Barrett's-related adenocarcinoma confined to the mucosa, endoscopic therapy is preferred over oesophagectomy or endoscopic surveillance (*Recommendation grade B*).

2. Endoscopic therapy of Barrett's neoplasia should be performed at centres where endoscopic and surgical options can be offered to patients (*Recommendation grade C*).

3. A minimum of 30 supervised cases of ER and 30 cases of endoscopic ablation should be performed to acquire competence in technical skills, management pathways and complications (*Recommendation grade C*).

4. ER should be performed in high-volume tertiary referral centres. Radiofrequency ablation (RFA) should be performed in centres equipped with ER facilities and expertise (*Recommendation grade C*).

ER for Barrett's-Related Neoplasia Associated with Visible Lesions

1**.** Endoscopic assessment will usually identify the area with the most advanced neoplasia. ER should aim to resect all visible abnormalities (*Recommendation grade C*).

2. ER is recommended as the most accurate staging intervention for Barrett's early neoplasia (*Recommendation grade B*).

3. ER should be considered the therapy of choice for dysplasia associated with visible lesions and T1a adenocarcinoma (*Recommendation grade B*).

4. For patients at high surgical risk, endoscopic therapy can be offered as an alternative to surgery for treatment of good prognosis T1b adenocarcinomas (T1b sm1, well differentiated and without lymph vascular invasion) (*Recommendation grade C*).

5. For T1b adenocarcinomas with involvement of the second submucosal layer or beyond (T1b sm2-sm3), endoscopic therapy should not be considered curative (*Recommendation grade B*).

6. The cap and snare technique with submucosal injection and the band ligation technique without submucosal injection are considered to be equally effective (*Recommendation grade A*).

Pathology Reporting of ER

1. Use of a minimum dataset for the reporting of ER specimens is recommended to ensure that all prognostic information is included in reports (*Recommendation grade C*).

2**.** The presence of tumour cells at the deep margin indicates incomplete resection and warrants further treatment (*Recommendation grade C*).

Imaging for HGD and T1 carcinoma: Role of CT–Positron Emission Tomography (PET) and Endoscopic Ultrasound (EUS)

1. Before ER, neither CT nor PET–CT have a clear role in the staging of patients with Barrett's HGD or suspected T1 cancer and neither is routinely required (*Recommendation grade B*).

2. Since EUS can both over stage and under stage T1 lesions, its routine use cannot be recommended for staging before ER for suspected early lesions (*Recommendation grade B*).

3. In selected cases where the endoscopies cannot exclude advanced stage on the basis of the endoscopic appearance of nodular lesions, EUS with or without fine needle aspiration (FNA) is recommended to inform the therapeutic decision (*Recommendation grade C*).

4. EUS with or without FNA of visible lymph nodes is recommended in selected cases with T1b (sm1) disease on staging ER for which endoscopic therapy is selected, because of the significant risk of lymph nodal involvement (*Recommendation grade C*).

Ablative Therapy for Flat HGD and Residual Barrett's After ER

1. In the presence of HGD or intra-mucosal cancer without visible lesions (flat HGD/intra-mucosal cancer), these should be managed with an endoscopic ablative technique (*Recommendation grade A*).

2. There are few comparative data among ablative techniques, but RFA currently has a better safety and side-effect profile and comparable efficacy (*Recommendation grade C*).

3. Eradication of residual Barrett's oesophagus after focal ER reduces the risk of metachronous neoplasia and is recommended (*Recommendation grade B*).

4. Endoscopic follow-up is recommended after endoscopic therapy of Barrett's neoplasia, with biopsies taken from the GOJ and within the extent of the previous Barrett's oesophagus (*Recommendation grade B*).

Surgical Management of Early Barrett's Neoplasia

1. Surgical therapy is considered the treatment of choice for early adenocarcinoma that has extended into submucosa because of the significant risk of lymph node metastasis (*Recommendation grade B*).

2. Oesophagectomy should be performed in high-volume centres, as these are associated with lower in-hospital mortality than low-volume centres (*Recommendation grade B*).

3. There is currently no evidence to support one technique of oesophago-gastrectomy over another. It is recommended that the procedure is tailored to the particular case and the expertise available in that centre (*Recommendation grade C*).

II. Endoscopic submucosal dissection (ESD): European Society of Gastrointestinal Endoscopy (ESGE) Guideline. Endoscopy 2015; 47:829-854

1 ESGE recommends endoscopic en bloc resection for superficial oesophageal squamous cell cancers (SCCs), excluding those with obvious submucosal involvement (strong recommendation, moderate quality evidence).

2. Endoscopic mucosal resection (EMR) may be considered in such lesions when they are smaller than 10mm if en bloc resection can be assured. However, ESGE recommends endoscopic submucosal dissection (ESD) as the first option, mainly to provide an en bloc resection with accurate pathology staging and to avoid missing important histological features (strong recommendation, moderate quality evidence).

2 ESGE recommends endoscopic resection with a curative intent for visible lesions in Barrett's oesophagus (strong recommendation, moderate quality evidence). ESD has not been shown to be superior to EMR for excision of mucosal cancer, and for that reason EMR should be preferred. ESD may be considered in selected cases, such as lesions larger than 15mm, poorly lifting tumours, and lesions at risk for submucosal invasion (strong recommendation, moderate quality evidence).

3 ESGE recommends endoscopic resection for the treatment of gastric superficial neoplastic lesions that possess a very low risk of lymph node metastasis (strong recommendation, high quality evidence). EMR is an acceptable option for lesions smaller than 10–15mmwith a very low probability of advanced histology (Paris 0-IIa). However, ESGE recommends ESD as treatment of choice for most gastric superficial neoplastic lesions (strong recommendation, moderate quality evidence).

4 ESGE states that the majority of colonic and rectal superficial lesions can be effectively removed in a curative way by standard polypectomy and/or by EMR (strong recommendation, moderate quality evidence). ESD can be considered for removal of colonic and rectal lesions with high suspicion of limited submucosal invasion that is based on two main criteria of depressed morphology and irregular or non-granular surface pattern, particularly if the lesions are larger than 20 mm; or ESD can be considered for colorectal lesions that otherwise cannot be optimally and radically removed by snare-based techniques (strong recommendation, moderate quality evidence).

III. Addendum to the British Society of Gastroenterology: Guidelines on the Diagnosis and Management of Barrett's Oesophagus (2015) https://www.bsg.org.uk/resource/bsg-guidelines-on-the-diagnosis- and-management-of-barrett-s-oesophagus.html.

IV. Revised British Society of Gastroenterology Recommendation on the Diagnosis and Management of Barrett's Oesophagus with Low-Grade Dysplasia (2017) https://www.bsg.org.uk/resource/bsg-guidelines- on-the-diagnosis-and-management-of-barrett-s-oesophagus.html

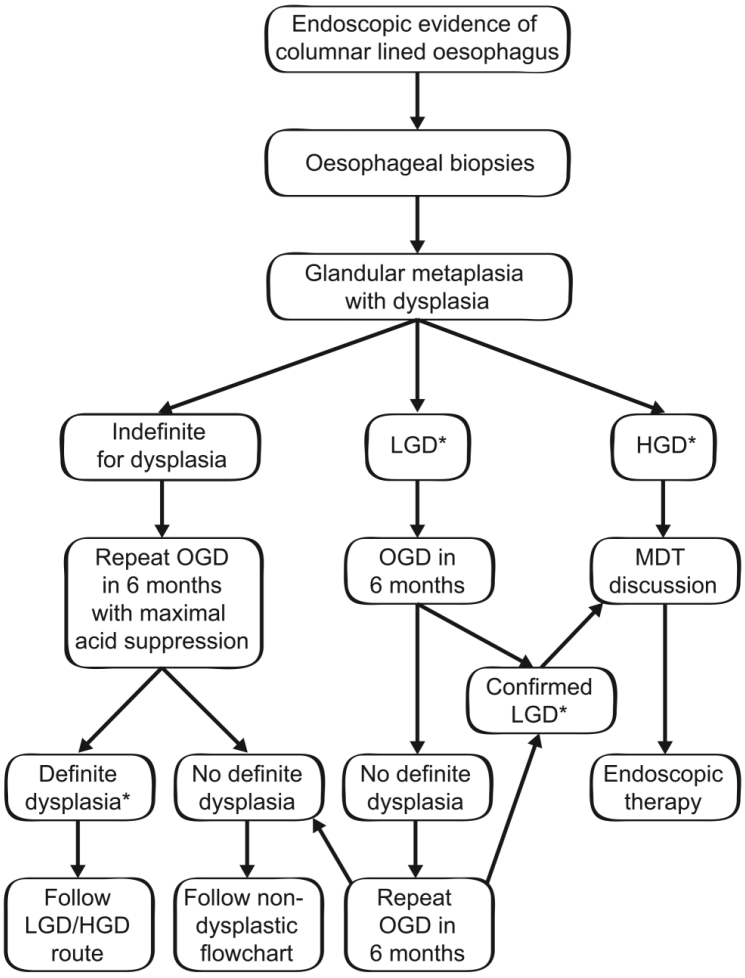
The most recent guidelines for the management of Barrett's oesophagus published in 2014 recommended endoscopic surveillance for patient with histological evidence of low-grade dysplasia (LGD) on random biopsies. In the last 2 years, new evidence on the natural history of LGD in Barrett's oesophagus and on the safety and efficacy of endoscopic treatment in this subgroup of patients has been published.

Duits *et al* have conducted a retrospective analysis of 293 patients with LGD diagnosed in community hospitals. Following consensus review, the original LGD diagnosis was confirmed in 27% of cases, while the remaining of the cases were downgraded to non-dysplastic Barrett's oesophagus or indefinite for dysplasia (IND). Patients with a LGD consensus diagnosis had a progression rate to high-grade dysplasia (HGD) or cancer of 9.1%/year over a median follow-up of 39 months. By contrast, patients whose diagnosis was down-staged to either non-dysplastic Barrett's oesophagus or IND had a conversion rate of 0.6% and 0.9%/year, respectively (evidence grade: low). This study reiterates the difficulty in making a pathological diagnosis of LGD, but also shows that confirmation by an expert pathologist from a different institution associates with a substantially higher risk of progression. In keeping with this study, a recent meta-analysis found that studies on cohorts with a low LGD/non-dysplastic Barrett's oesophagus ratios (<0.15), indicative of a more stringent and robust dysplasia diagnosis, reported a significantly higher annual incidence of cancer (0.76%, 95% CI 0.45% to 1.07%) compared with studies with a ratio >0.15, where many LGD cases were likely over diagnosed (0.32%; 95% CI 0.07% to 0.58%) (evidence grade: low).[3]

With regard to endoscopic treatment, a recent multicentre randomized controlled trial compared the outcome of 68 patients with LGD treated with radiofrequency ablation (RFA) with an equal number of patients undergoing annual endoscopic surveillance.[4] The main inclusion criterion was a diagnosis of LGD confirmed by a central pathologist with extensive experience in Barrett's oesophagus. Over a 3-year follow-up period, 1% of patients in the treatment arm progressed to HGD or cancer, compared with 26.5% in the control arm ($p<0.001$) (evidence grade: high). Complete eradication of dysplasia and intestinal metaplasia by RFA was achieved in 98% and 90%, respectively. The most common complication was stricture, which occurred in 12% of patients, but this was successfully managed in all patients with endoscopic dilatation.

Taken together, the new published data suggest that a consensus diagnosis of LGD by independent pathologists correlates with a significant risk of progression to HGD/cancer and that RFA significantly reduces this risk. This strongly indicates that endoscopic ablation, preferably with RFA, is an appropriate treatment for Barrett's oesophagus with LGD. Due to the considerable diagnostic difficulties, LGD should be diagnosed and confirmed by an expert GI pathologist in at least two endoscopes. Treatment options should be discussed by an upper GI multidisciplinary team (MDT) to ensure pathology review and assess patient fitness and endoscopic therapy should be restricted to high volume centres as per main 2014 guidelines. As noted in the current guidelines, p53 immunohistochemistry can be a useful diagnostic adjunct.

Therefore, the new recommendation on the management of LGD in Barrett's oesophagus is: Patients with LGD should have a repeat endoscopy in 6 months' time. If LGD is found in any of the follow-up oesophago-gastro-duodenoscopy and is confirmed by an expert GI pathologist in at least two sets of biopsies, the patient should be offered endoscopic ablation therapy, preferably with RFA, after review by the specialist MDT. If ablation is not undertaken, 6-monthly surveillance is recommended (recommendation grade A for endoscopic therapy and C for surveillance).



* dysplasia needs to be confirmed by 2 independent GI pathologists

Summary and Comments:

To summarise, RFA is recommended for LGD in BO and in flat HGD. With regards to EMR and ESD, this depends on what you are doing it for; if this for a nodular area in BO, then the cancer standards must apply, so EMR is perfectly acceptable in superficial oesophageal lesions of less than 15 mm maximum diameter when a complete en bloc removal can be achieved. To remove an oesophageal cancerous or a possible cancerous lesion in a piece meal manner is a direct breach of our learnt surgical oncological principles which is what happens when lesions larger 15 mm are removed by EMR. I put it to you, not being able to do ESD or being less able at accomplishing it as good as EMR is not a good enough reason to choose it, as this is done in the very cohort of patients are set to potentially benefit the most from a sound complete en bloc endoscopic removal of their early cancer.

And in terms of evidence, the frequent quote of "*lack of evidence of benefit from ESD*" compared to EMR is not an evidence of lack of benefit; furthermore, western experience is gathering to support safety of ESD. Alex Chen from McGill University, Montreal, QC, Canada presented their experience at DDW in San Diego last month of their 93 consecutive ESD for oesophageal and gastric neoplasia and the message is very clear; endoscopic submucosal dissection is a viable, effective, and safe option for superficial lesions of the stomach and oesophagus. There is an urgent need to upskill and efforts should be made to identify and address barriers to adoption and dissemination of this technique in the UK and Professor Pradeep Bhandari's efforts are to be commended for championing this in the UK. Furthermore, the current UK guidelines are outdated from ESD view point and needs revisiting to reflect the Western experience.
It is my recommendation that UGI cancer surgeons should acquire such advanced endoscopic skills to enable delivery of the full complement of treatment options to their patients.

2019 Gloucestershire Upper GI Symposium (GUSS):

GUSS took place on the 16th and 17th of May; the programme was inclusive of practical topics of interest, the content was informative and the speakers engaged the audience sparking interesting discussion during the generous Q&A sessions. It was an excellent educational activity and I would recommend you to save the date for the future symposia and I congratulate Mr Shameen Jaunoo for such a successful meeting. All lectures were excellent especially Prof Hugh Barr one titled "Management & Imaging in Barrett's Oesophagus".

Professor Barr captured the audience with his usual down to earth, entertaining but very informative style. He updated the audience of the advances in matters relating to Barrett's oesophagus during his memorable talk. This was followed by a another excellent lecture by Mr Simon Dwerryhouse, Consultant Upper GI Surgeon who touched on the importance of training in advanced therapeutic endoscopies for UGI surgeons and that such training opportunities are now available in European centres. Mr Nick Maynard, AUGIS President Elect, provided a comprehensive review of management of achalasia and highlighted need for centralisation of such services.

Cancer Research UK - International Oesophageal Symposium Cancer 2019

Convener: Professor Rebecca Fitzgerald

The International Symposium on oesophageal cancer has now become a bi-annual event and this year's conference was held in London at the Royal Institution which was an inspiring venue. The audience was truly international and represented a diverse range of interests and included students as well as senior scientists and clinicians from academia and industry. The conference started with a thought provoking talk from Imran Haque entitled "We are legion". Imran is not from the Oesophageal cancer field but he lived up to his reputation and provoked the audience to think outside the box and considered the merits of a mechanistic versus an empirical approach for making new scientific discoveries. He also introduced us to the interactive Q&A tool called slide which proved to be a great way of engaging with the audience throughout the conference which had lots of time for discussion built into the schedule. Over the two days we covered: epidemiology including the African perspective; a session on cellular molecular drivers of the disease; how to build a more robust translational pipeline; development of new therapies; the tumour microenvironment and last but not least early detection and prevention. A highlight was the future leaders' session which brimmed with enthusiasm and there was much discussion over the posters especially at the drinks reception. I think that everyone would agree that it was a highly inspiring and stimulating event which brought new perspectives from outside as well as from within the field and the patient perspective was a stark reminder of the importance of research into this disease. We are very grateful to Cancer Research UK for their support - without which this symposium would not have been possible.

I do you hope that you find the contents of this newsletter helpful, it is worth mentioning that the comments section represent my own views and not a formal AUGIS stance. Finally, I wish you all a lovely summer.

Ashraf Rasheed

References;
1 Jankowski JA. Barrett esophagus and surveillance in the United Kingdom. Gastroenterol Hepatol. 2009;5(11):766-8
2 Jankowski J, Barr H, Wang K, Delaney B. Diagnosis and management of Barrett's oesophagus. BMJ. 2010;341:c4551
3 Wild CP, Hardie LJ. Reflux, Barrett's oesophagus and adenocarcinoma: burning questions. Nat Rev Cancer. 2003;3(9):676-84
4 DynaMed. Barrett esophagus. Ipswich (MA): EBSCO Publishing; 2012.
5 Sikkema M, de Jonge PJ, Steyerberg EW, Kuipers EJ. Risk of esophageal adenocarcinoma and mortality in patients with Barrett's esophagus: a systematic review and meta-analysis. Clin Gastroenterol Hepatol. 2010;8(3):235-44