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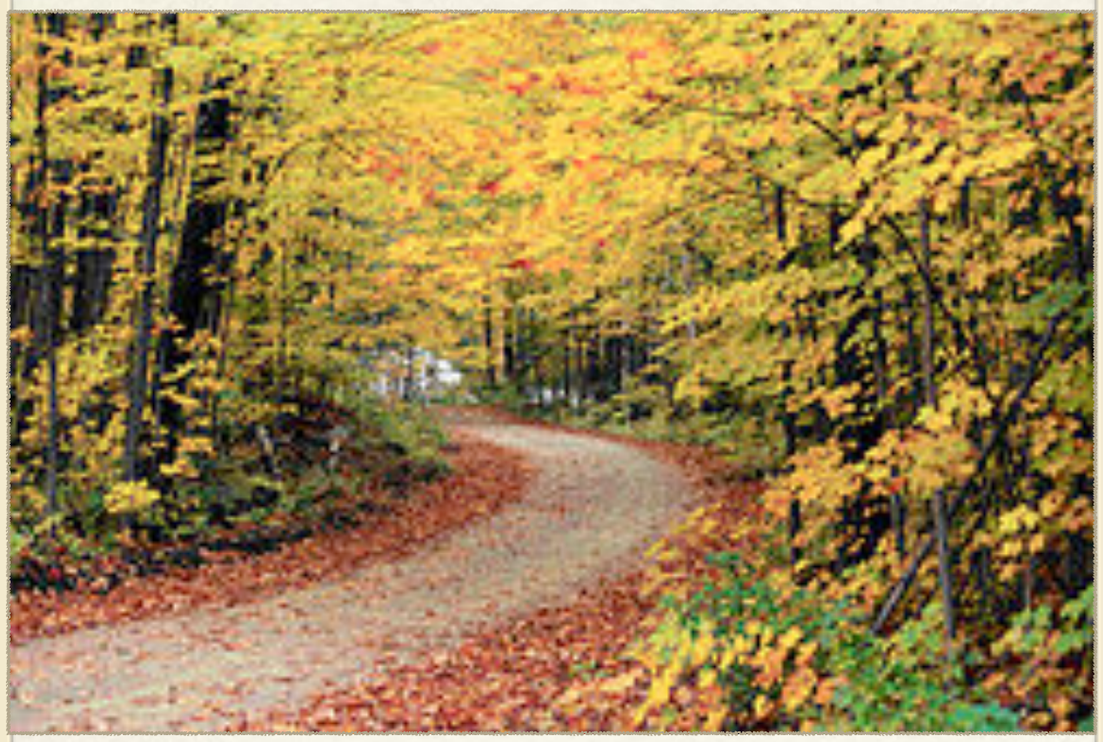

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Editorial

Welcome to the the Autumn Issue of the JPCSG.

I hope the summer went well for you all and , from all accounts, the recent ASM was a great success with lots of great feedback. So I only have one hypocritical thing (I couldn't make it either!) to say to those of who weren't there.....why not? The endoscopy meeting will be in the New Year and we will have another rip roaring ASM last year so come along!

In this issue, we have a pot-pourri of articles ranging from GORD, a review of the presentations from the recent UEGW, a case history from the ever dependable Mike Cohen and a personal diatribe from me regarding inter consultant referrals.

Finally, as some of you may well know, I will not be nagging you in the future regarding contributions to the journal and nor will I plague you with my eccentric ramblings. I have decided to step down from the roles of journal editor, and also Secretary of the Society. Watch this space for a far more professional journal and I wish my successors all the best!

It's been fun, but as my mother used to say, when one door closes... always have a spare key. No, I never knew what that meant either.

John O'Malley, Editor.

INTRODUCTION

What do and should we do with GORD not responding to conventional treatments and doses? I am indebted to Daniel Sifrim and Philip Woodland for contributing this article.

In their article, which will be of interest to all in primary care, they outline the problems of refractory GORD and some of the answers.

SECTION 2

Refractory Gastro-Oesophageal Reflux Disease

Daniel Sifrim and Philip Woodland from Barts and The London School of Medicine and Dentistry , Queen Mary University of London

Introduction

Gastroesophageal reflux disease (GORD) can be defined as "a condition which develops when the reflux of stomach contents causes troublesome symptoms and/or complications"[1]. It has a global impact on health and quality of life, affecting a substantial proportion of the world's population[2]. Whilst over recent decades there have undoubtedly been important and successful advances in the treatment of GORD, the prevalence of the disease appears to be increasing. When defined as the presence of at least weekly heartburn or regurgitation, there is an estimated prevalence of 10-20% in Western Europe and North America[3].

Acid suppression with PPI therapy is the mainstay of treatment for GORD in the majority of symptomatic subjects. With such a high prevalence of GORD in the community, a small proportion of patients who do not respond adequately to PPU therapy would be deemed a significant clinical problem. Indeed, it has been documented that 10 - 40% of patients with suspected GORD do not respond to PPI therapy[4].

What is refractory GORD?

In research studies, "incomplete" response to PPI therapy is usually defined as less than 50% improvement in the main symptom (which must be reflux-related) after 12 weeks of PPI therapy. Of course, in real life practice the judgement on whether treatment response has been satisfactory is most often made by the patient. This is particularly true of a disease like GORD where the endpoint of treatment is almost entirely symptomatic (unlike, for example, inflammatory bowel disease where mucosal healing is also often considered). Thus, the definition of refractory GORD is often subjective and patient expectations may be influenced by age, cultural background, comorbidity and social status[5].

Most patients are considered refractory to PPI therapy when the response remains inadequate after the PPI dose has been escalated to twice daily. Approximately 25% of those patients with inadequate response to once-daily PPI will gain benefit from an increase to twice-daily[6].

Noting the above, perhaps a pragmatic definition of refractory GORD could be ongoing troublesome symptoms of GORD despite 3 months of double-dose PPI therapy.

Why is the patient refractory to PPI therapy?

1. Are the symptoms suggestive of reflux, or an alternative diagnosis?

The first thing to consider if the patient is refractory to PPI therapy is whether the symptoms are indeed of reflux disease. Many symptoms are sometimes erroneously attributed to reflux, when the aetiology is quite different. For example, some patients diagnosed with GORD actually have symptoms of dyspepsia. The Montreal Classification of reflux defines symptomatic oesophageal reflux syndromes as either typical reflux syndrome, or reflux chest pain syndrome. Typical reflux syndrome is the most well-established symptom association with GORD, comprising of the

so-called “typical symptoms”: heartburn and regurgitation. Heartburn is a retrosternal burning sensation, and regurgitation is the detection of refluxed gastric content into the mouth or hypopharynx. Although heartburn is not specific for GORD, results of studies using acid suppression therapies for treatment of heartburn provide indirect evidence that GORD is the most common cause of heartburn6. When heartburn and regurgitation are present as the only symptoms, they are specific but not sensitive in the diagnosis of GORD[7]. Typical reflux symptoms are characteristically worsened after eating, on bending, and on lying down (especially on the right side).

In chest pain reflux syndrome there are episodes of non-heartburn chest pain caused by gastroesophageal reflux. The pain can sometimes be indistinguishable from cardiac chest pain.

Outside of these oesophageal syndromes, symptoms are less likely to be caused by reflux. A on detailed questioning it can sometimes be ascertained that whilst typical reflux symptoms have improved with PPI therapy, the “refractory” symptoms are in fact dyspepsia or globus, for example, and no amount of PPI dose escalation will improve these.

Rarely, heartburn can be due to other disease such as eosinophilic oesophagitis or severe oesophageal motility disorders such as achalasia (in both cases a history of dysphagia is important).

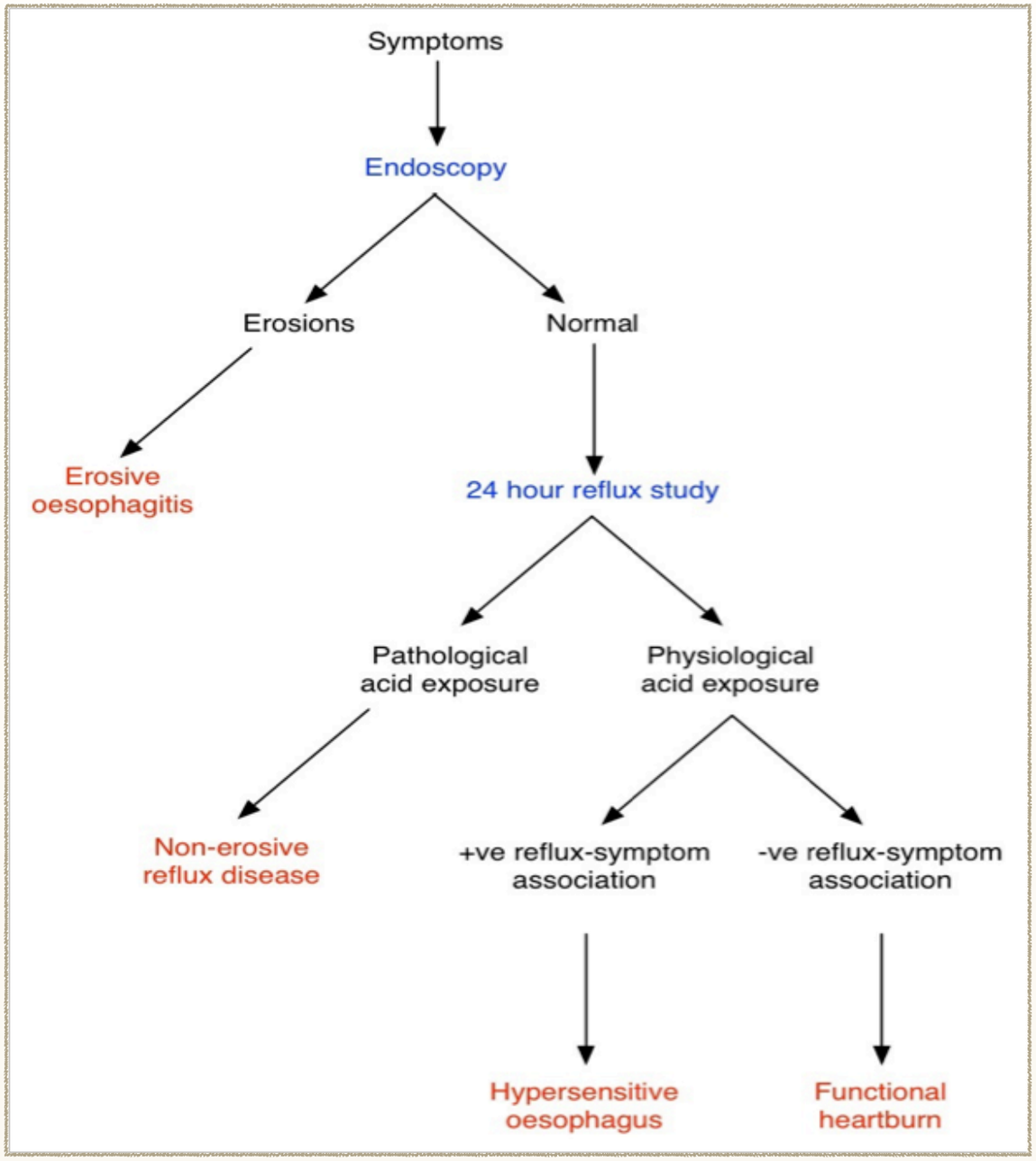
2. Are the symptoms due to functional heartburn?

There are a significant proportion of patients who present with typical reflux symptoms, but do not have these symptoms due to reflux disease. This group of patients (with normal endoscopy, normal oesophageal acid exposure, and no reflux-symptom temporal association) are defined as having functional heartburn. Due to the fact that functional heartburn is not caused by gastro-oesophageal reflux, patients with the disorder do not respond to PPI (in fact PPI response precludes a diagnosis of functional heartburn). Patients with functional heartburn account for approximately 30% of patients presenting with GORD-like symptoms. They are more likely to present with functional overlap symptoms of dyspepsia (fullness, early satiety, bloating and nausea) and irritable bowel syndrome than patients with true GORD[8]. Patients with functional heartburn are also more likely to have comorbid psychopathology than patients with true GORD[9].

Functional heartburn may be distinguished from GORD on some occasions by endoscopy, but it should be noted only about 30% of patients with GORD have macroscopic erosions (the others have so-called non-erosive reflux, or NERD). As such, the definitive method to distinguish the two is by 24-hour reflux monitoring, either with pH or combined pH-impedance studies. This can be used to assess oesophageal acid exposure and the statistical association between reflux events and symptoms. A diagnostic algorithm is shown on the next page (figure 1).

Note also the presence of the diagnosis of hypersensitive oesophagus on figure 1. This is defined where there is negative endoscopy, normal oesophageal acid exposure on 24-hour monitoring, but a significant temporal association between reflux events and recorded symptoms during the 24-hour monitoring. Since the symptoms are due to gastro-oesophageal reflux, patients with hypersensitive oesophagus fall under the GORD umbrella rather than functional heartburn, but sometimes a different treatment strategy to other GORD patients is required (see below).

Figure 1: Diagnostic algorithm for GORD symptoms



3. Why are patients with true GORD refractory to PPI therapy?

When patients with dyspepsia and functional heartburn are excluded, there still remain a significant proportion (around 20%) of patients with true GORD who are refractory to PPI. There are several possible mechanisms for this, several with therapeutic implications.

a. Residual acid reflux.

Probably the most important mechanism for symptoms due to residual acid reflux is non-compliance with medications. In particular there are patients who have persistent symptoms because of an intermittent, "reactive" approach to PPI taking. Of course, this is reasonable if the patient is content with this (if there are no macroscopic complications of GORD), but it should be emphasised that some cases of GORD require continuous dosing of PPI in the absence of symptoms, as a preventative measure.

The next most important reason for residual acid reflux is probably incorrect timing of dosage. It is important to ensure that PPIs are taken 30 minutes before meals to ensure their activity at the time of maximum proton pump activity.

Outside of conditions of poor compliance and incorrect PPI timing the role of residual acid exposure in refractory GORD remains controversial. Whereas it appears to play a role in patients taking only once-daily PPI, it is likely to be of much less importance in patients taking twice-daily PPI therapy ((in one study 38% of patients taking once-daily PPI had abnormal 24-hour oesophageal acid exposure, but only 4% in those on twice-daily PPI). Indeed a study by Gasiorowska et al demonstrated that the amount of residual acid reflux was not different in responders to PPI and non-responders[10].

A very small number of patients may have excessive residual acid exposure due to rapid PPI metabolism, or a hypersecretory condition such as Zollinger-Ellison syndrome.

b. Symptoms due to weakly acidic reflux and duodeno-gastro-oesophageal reflux.

Not all gastro-oesophageal reflux is acidic, particularly in the "on" PPI condition, where most reflux is in the "weakly acidic" range of pH 4-6. Data from GORD and NERD patients has shown that 30% of symptoms may be associated with reflux episodes with a pH of 4-7 [6, 11-13]. Emerenziani et al. also showed that

although most symptoms were related to acid, NERD patients in particular were sensitive to weakly acidic reflux events (accounting for 24% of all their symptoms) [14]. Obviously such symptomatic reflux will not be responsive to PPI therapy.

The refluxate consists of not just acid, but other components of gastric juice including pepsin, and elements of duodeno-gastro-oesophageal reflux (DGOR).

Reflux of duodenal contents into the oesophagus has been hypothesised to cause damage due to the toxic effects of components such as bile acids and pancreatic enzymes [15]. Most DGOR, however, occurs in combination with acid, and PPI therapy appears to reduce both acid and bile reflux[16]. Studies have implicated DGOR ashaving a potential role in refractory reflux disease, but have suggested that this role is small, being associated with less than 10% of symptom events[10,17].

a. Oesophageal hypersensitivity.

Visceral hypersensitivity is an increasingly recognised cause of gastrointestinal symptoms, including reflux symptoms. Oesophageal hyperalgesia and/or allodynia are likely to play a role in functional heartburn, but also to a variable degree in GORD. Experimental data suggests that patients with functional heartburn and NERD are more sensitive to oesophageal acid challenge, balloon distension or electrical stimulation than patients with erosive oesophagitis or controls[18]. The cause of this sensitisation is unclear, but may include changes in mucosal barrier integrity, microscopic mucosal inflammation, upregulation of mucosal acid-sensitive receptors and sensitisation of peripheral afferent nerves (peripheral sensitisation), and/or sensitisation of the spinal dorsal horn neurons (central sensitisation)[19].

b. Psychological comorbidity.

It has been shown that patients who respond less well to PPI therapy were more likely to have concomitant psychological distress[20]. Often it is reported by patients that GORD symptoms are exacerbated by psychological stress, and experimental stress increases oesophageal sensitivity to stimulus[21]. It is most likely that psychological stress contributes to central sensitisation mechanisms, although some animal data suggests there may also be a sensitising effect peripherally[22].

How to investigate the refractory GORD patient

It follows that the investigation of the refractory patient is driven by the factors outlined in the section above. A careful history will elucidate those patients with an alternative diagnosis, and (commonly) those patients in whom heartburn has improved on PPI therapy, but the "refractory" symptoms are related to another symptom set such as dyspepsia.

Concomitant functional disorders and psychological comorbidity may suggest a diagnosis of functional heartburn. PPI dosing and timing should be interrogated.

Some patients will require further testing. Endoscopy may show the presence of oesophageal erosions or Barrett's oesophagus, may help rule out less common causes of heartburn such as eosinophilic oesophagitis, or may suggest severe oesophageal motility disorders. In some cases it can offer much needed reassurance to the patient.

In difficult cases 24-hour ambulatory reflux monitoring can be invaluable. Measurement of oesophageal acid exposure and temporal symptom-reflux correlation "off" PPI can distinguish true GORD from functional heartburn. In patients with known reflux disease, the addition of impedance measurements to an "on" PPI study can detect symptoms due to weakly acidic or alkaline reflux events. Equally usefully, it can help distinguish those symptoms that are due to ongoing reflux, and those that are due to other causes.

Treatment of the refractory GORD patient

Naturally, treatment will be guided by the likely cause of the refractory symptoms.

In all cases lifestyle modification may be of benefit. Weight loss and avoiding late-night meals can be effective interventions for GORD[23]. Smoking cessation is also likely to be beneficial.

Antisecretory therapies

As detailed above, the most useful therapeutic intervention in refractory patients taking once-daily PPI may be a trial of twice-daily PPI. This is based on the additional gain in controlling acid exposure. Clinical trial data supporting this is sparse, although studies have shown that a switching single dose lansoprazole to twice daily lansoprazole or double dose omeprazole or esomeprazole can achieve adequate symptom control in 20-30% of patients after 6-8 weeks[24,25].

The addition of H2-antagonists at night is a common therapeutic intervention, and it is known to reduce nocturnal acid breakthrough even in patients on twice-daily PPI25. However clinical data supporting this is lacking, and persistent use is associated with rapid loss of efficacy. As such these drugs are probably best employed on an intermittent, on-demand basis.

Pain modulators

In patients with functional heartburn, drugs targeting visceral hypersensitivity are appropriate. The same is likely to be true in patients with acid sensitive (hypersensitive) oesophagus not responding to PPI. Drugs such as tricyclic antidepressants, SSRIs and SNRIs confer a visceral analgesic effect by acting at the central nervous system and/or sensory afferent level. Citalopram 20mg daily has been shown to be beneficial in patients with acid hypersensitive oesophagus and refractory symptoms[26]. In clinical practice low dose amitriptyline can also be effective, and there is some evidence to suggest the benefit of venlafaxine helps in functional chest pain disorders[27].

Anti-reflux therapies

In those patients with symptoms thought to be due to ongoing reflux (e.g. weak acid or DGOR) despite PPI therapy, options to reduce reflux events can be considered.

Antireflux surgery is a very effective treatment for controlling reflux. In patients responding to PPI therapy, antireflux surgery usually offers a durable symptom relief. However, in patients refractory to PPI therapy the outcome is less good, with persistent symptoms "on" PPI a factor associated with an unfavourable outcome from surgery[28]. This may be in part due to patient selection. It would follow that in order to consider patients for antireflux surgery, a very careful phenotyping process should be undertaken. Certainly an abnormal pH study "off" PPI therapy appears important for a good outcome, although recent data suggests that long-term outcome is not always satisfactory even in those patients who partially respond to PPI therapy. Perhaps we can be most confident in referring only those patients with a positive reflux-symptom association during pH-impedance monitoring "on" PPI therapy: i.e. those in whom non-acidic reflux events appear to be clearly causing symptoms. Some data suggests that good post-operative outcomes can be achieved in these situations[29], although good controlled outcome data is lacking.

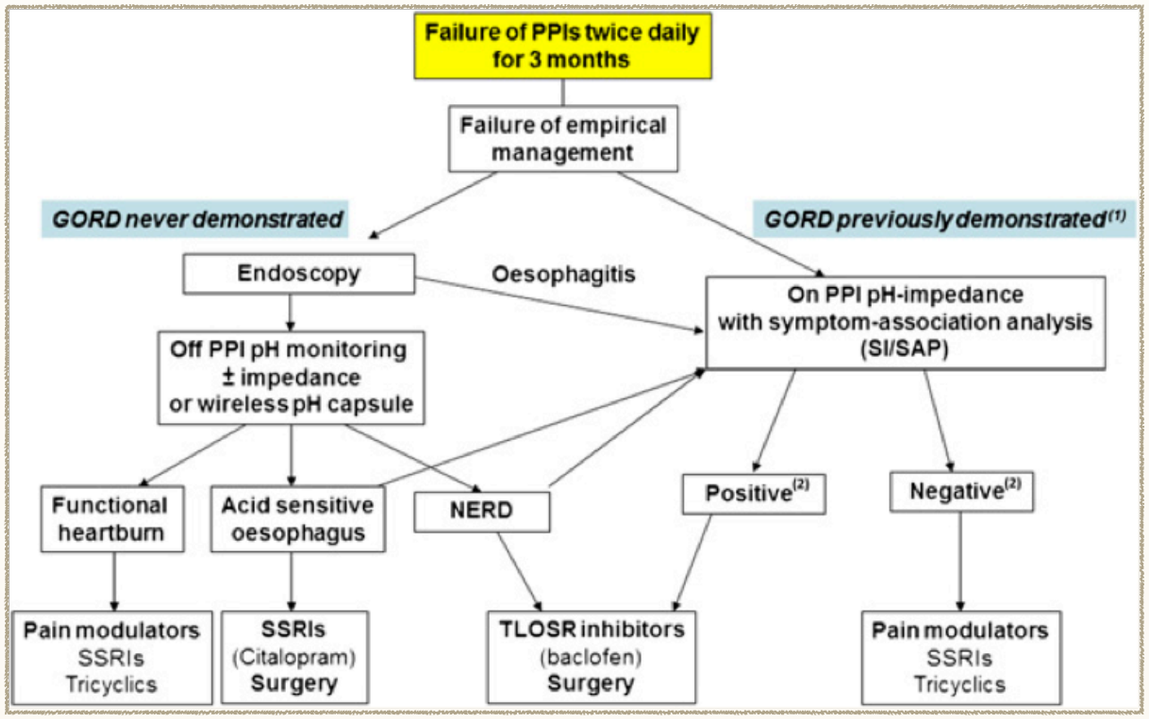
A medical approach to reflux control exists, and has been an area of intense research in the past decade. Transient lower oesophageal sphincter relaxations are the most common mechanism for reflux events, and drugs that reduce the frequency of these relaxations are therefore desirable. The GABAB agonist baclofen is such a drug, and 10mg three times per day can reduce acid reflux events and reflux-related symptoms[30]. It can therefore be an option in refractory reflux disease, and is sometimes used in specialist clinics. Unfortunately its use is limited by a poor side-effect profile. Development of newer, less toxic, GABAB agonists has now been halted predominantly to insufficient clinical efficacy.

Conclusion

The management of refractory GORD symptoms remains a significant clinical burden, and can pose a difficult clinical challenge.

Perhaps the most important step in managing these patients is to establish the correct diagnosis, and to establish which patients truly have refractory GORD. In these patients clinical benefit may be gained by aggressive anti-secretory therapy or even anti-reflux procedures. In others reassurance and the use of pain modulatory drugs is likely to lead to a more satisfactory clinical conclusion than further attempts to reduce oesophageal acid exposure.

Figure 2: Suggested approach to PPI refractory GORD



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SECTION 3

The Retrospectoscope- The most accurate diagnostic tool we have

Mike Cohen, GP and Endoscopist, Westbury on Trym,Bristol.

INTRODUCTION

- *A curious case history this, which raises lots of questions and teaches several valuable points. Mike Cohen reflects, to his great credit,with great honest ,on this interesting case which shows that we can all be experts on looking back but the challenge of primary care is to, often, rely on your skills rather than investigations.*

A 71 year old with biopsy proven coeliac disease presented to her GP in August 2007 with diarrhoea. She thought she must have inadvertently ingested gluten when she dined out. Her symptoms were nausea and watery diarrhoea. Stool culture was normal. Over the next 6 weeks she lost 17lbs in weight and was re-referred to a consultant gastroenterologist. Clinical examination was normal. He thought it sounded like food poisoning but wondered about giardiasis.

He arranged for a further duodenal biopsy and blood tests.

The duodenal biopsy was reported as being consistent with unresponsive coeliac disease. It was noted also that her LFT's were very abnormal: -

Alkaline Phosphatase 227 (NR 20-110)

ALT 138 (NR 5-40)

Albumin 27 (NR 35-40)

CRP 7(NR 0-7.5)

Hb 10.2 with normal folate and ferritin levels.

In the past she had suffered with iron deficiency anaemia which led originally to her diagnosis of coeliac disease.Blood testing also revealed positive smooth muscle antibodies.

Ultrasound of her liver was non contributory and a CT scan of her abdomen was normal-there was concern about a possible lymphoma in view of her anaemia and weight loss.

Liver biopsy was reported as showing mild inflammation with a little excess fat.

The conclusion here was that this lady has unresponsive coeliac disease and autoimmune hepatitis.

She was started on prednisolone 30mg daily and started to feel much better. She was reviewed in clinic 2 months. Her LFT's had improved with her ALT coming down to 50 and her steroids were reduced. She was started on azathioprine.

However 3 months later she became iron deficient- Hb 9.9.

In February 2008 she developed RUQ pain and looked very unwell. She felt sick and was not eating. Is suspected a flare up of here autoimmune hepatitis. Her LFT's had deteriorated: -

Alkaline Phosphatase 187 ALT 109 Albumin 34

She was re referred to her consultant who started to do some tests and considered investigating her anaemia.

However in May 2008 she developed constipation and over a fortnight this progressed. She was admitted as an emergency with large bowel obstruction

She was taken to theatre where a colectomy, bilateral oophorectomy and appendicectomy were performed for metastatic carcinoma of the sigmoid colon.

She was referred to oncology and decided to opt for palliative chemotherapy. Treatment with 5FU and oxiplatin was started. She developed a gram negative septicaemia during the third cycle. She was referred to the palliative care team and subsequently gradually became weaker with increasing anorexia, oedema and jaundice. She died peacefully at home on 17 April 2012.

Discussion

The retrospectoscope is the most accurate diagnostic tool we have. It is interesting that her first thought when she developed diarrhoea was that she must have inadvertently eaten some gluten. She may of course have had 3 separate pathologies but I do wonder now whether this lady had colonic cancer from the outset. Her initial symptoms were watery diarrhoea and weight loss. This points to colonic pathology but her consultant commented that a small bowel problem was the most likely cause for her symptoms. The CT scan of her abdomen was unremarkable and there certainly was no radiological evidence of liver metastases when she first presented. Just before a definitive diagnosis was made she became profoundly constipated. According to 2 week wait colorectal cancer guidelines a change in bowel habit especially towards looser stools is a red flag but not constipation. I wonder whether these guidelines need revising. I have recently scoped a patient with recent onset constipation who had a large obstructing rectosigmoid cancer.

This patient was a well informed highly intelligent person. She had in the past been a nurse. She was shocked when a diagnosis of metastatic colorectal cancer was made. I certainty felt very uncomfortable visiting her after her operation to see how she was and to discuss her on going management. I wondered whether I had been too passive in her management. She was under a consultant and I had left all the thinking to him. Was that right? We discussed her presentation and she was accepting but very disappointed. We both agreed she should have had lower GI investigations earlier.

A difficult, complex and ultimately very sad case. I have learned a lot.

INTRODUCTION

❖ *The following is a distillation of some of the bits I got from the recent UEGW endoscopy abstracts and posters that made me say*

'That's interesting!'

I know.....I am a geek.

UEGW- the best bits.

John O'Malley JPCSG Editor

- **The small polyp question.**

♣ Should one be worried about small (<5mm) polyps seen on colonoscopy? Caroli et al. looked at the current resect and discard strategy. It is estimated that there is a prevalence of between 1.7% and 5.9% of advanced adenomas seen in such polyps so is the present policy advisable? Their work showed that it was not economically sound to send all small polyps for histology as the risk was so small (5.4%).

- **Taste and GORD**

♣ It has been known for a while that many patients with GORD have altered taste sensation. This study by Verlezza et al suggested the

ability of GORD patients to score acidity better than controls may be due to the possible sensitisation of sour taste receptors. It may also explain why they often find food lacking taste and needing more salt.

- **Coeliac Disease and depression**

♣ I know I do have an obsession about looking for coeliac disease everywhere but it continues to be linked to many other conditions. Tortora et al found that post partum depression was far more common in CD patients than other groups even if the patients were following a gluten free diet. They suggest such patients should be screened earlier than usual and that undiagnosed CD should be looked for in those with PPD.

- **Coeliac Disease part 2.**

♣ Gabrielli et al showed that, in Italy at least, a strong association between psoriasis and CD. This primary care study reflected for the first time similar results seen in secondary care and is part of an ongoing trail to see if a gluten free diet could help psoriasis in those who are serologically positive.

- **Colonoscopies; the aftermath**

♣ What happens to patients after colonoscopy? This study by Stevens et al based at the West Middlesex University Hospital audited the morbidity and healthcare costs after outpatient colonoscopy. Out of the 1115 scopes studied, 22 visited the A&E afterwards within 14 days with 14 being procedure related. 5 needed admission with abdominal pain being the commonest cause and bleeding, sedation and post polypectomy problems making up the rest. The average cost for the A&E attendance was £145 with those needing admission costing £3338 with total cost for all studied of £18720 over the 12 month period.

INTRODUCTION

- *Finally, if I going to go, I might as well leave with some controversy. This is a personal piece based on my experiences of being a open access endoscopist and the restriction we, along with our secondary care colleagues, are being put under regarding C2C or consultant to consultant referrals. I would be delighted to hear your views!*

SECTION 5

The ethics (or lack of) regarding restricting referrals

John O'Malley, Medical Director, Mastercall Healthcare, Stockport and Hospital Practitioner, Wirral

We live in difficult times and health care provision has not been spared in this age of austerity[1]. What makes primary care in the UK so different is the intimate and integral nature of the GP contract with the State and it's new (or some would say old but more intensified) role in commissioning. With the purse strings getting ever tighter, commissioners are increasingly looking for 'quick wins', changes that will give the highest return in the shortest of times. Consultant to consultant referrals or C2C referrals are an obvious target.

I must first declare that any opinions put forward in this article are mine alone and do not form a declaration of PCSG or Mastercall policy . So I don't go completely

mad, I do an open access endoscopy clinic on a Wednesday evening for the hospital local to my home. Recently, the Trust, after discussions with the local commissioners, have told all open access endoscopists not to make referrals based on our endoscopic findings. This applied not only Barrett's clinic referrals but also to follow up endoscopy for gastric ulcers etc. It did not apply to referrals relating to confirmed or suspected malignancy. We, therefore, now have to 'ask' the referring GP to refer for the follow up referral or endoscopy.

I have several problems with this policy but the main one relates to patient safety. I was a full time GP principal for 16 years before I moved into medical management and I still do GP sessions now for our practice. I am well aware of the mountain of information that descends on a practice each day, some of which adds very little to the information we need for patient care. Many are very detailed and I think it would be unusual to hear that a GP has not once just scanned a letter for relevant information. So there is potential for data to be missed, not entered on databases and, therefore, forgotten.

Is there any evidence for this? Worldwide evidence shows that, despite our best efforts, important information is missing from many consultations and such omissions do have an effect on patient care. Smith found in the U.S. That 13.6% of primary care consultations had missing clinical information leading to adverse patient outcomes in about a half of all cases[2]. Looking at it from another angle, one study looking at 344 cases of medical errors had, as the main cause, the unavailability of information in nearly 10% of cases[3]. Finally, in Australia, Wilson found that 1.8% of 2353 adverse events were due to the physician not being aware of information with 26.4% of these cases leading to permanent disability[4].

Are C2C referrals a big problem? I am not convinced there is enough evidence that secondary care is referral crazy and referring 'just for the sake of it'. Trawling through the papers , there is very little evidence that widespread inappropriate referring is going on and although, yes, specialist clinics do refer more, these referrals are usually appropriate and in the patients' best interests[5]. It certainly is becoming a big issue for primary care and especially in their commissioning role as reflected by DoH figures reported as showing a 9% year on year increase for the first quarter of 2012/13. There is also evidence that as referrals by GPs is falling in some areas due to, for example, peer

review schemes, C2C referrals are rising. A report by CHKS showed that over a space of five years GP referrals had fallen from 69% to 62% but C2C referrals had risen from 16% to 21%[6]. But this does need to be seen in context. Looking at the figures closely, one sees that oncology had the highest number.

NHS Oxfordshire is leading the way with electronic systems being set up to allow GPs to receive copies of all non emergency C2C referrals but it is not clear whether they are empowered to stop them. I think the copying of such letters is only right as the GP certainly has the right to know what is happening to their patient but I was surprised to find that an audit in NHS Rotherham showed that GPs were unaware of such referrals occurring in 42% of cases and that in 7% of cases the reason for such a referral was unclear [7]. Other schemes have more teeth with Newcastle and Rotherham CCGs introducing protocols demanding that consultant ask the permission of GPs before referring to colleagues. Dr Mike Dixon of NHS Alliance has been quoted as saying it is the single biggest cause of rising referrals but doesn't say whether it is appropriate or not[8]. In fact, he confirms this by, in the same quote, saying that 'In most cases , it is bonafide. But there are suspicions (my italics) in some hospitals that this is fuelling an industry in payment by result tariffs'. Where is the evidence for this? Many C2C referrals in gastroenterology are to tertiary centres for further advice to surgical colleagues with expertise in a certain field. Would any local GP know who was the best person to refer surgical cases of IBD to? Where would we get this information? I can, honestly, say in my 19 years as a hospital practitioner in gastroenterology I have neither initiated or witnessed an unnecessary C2C referral and most have resulted in a definite improvement in patient care. For example, I have seen many cases of endometriosis, either misdiagnosed or accompanying IBS, which I have referred to gynaecologists. Surely I have an ethical duty to give the best care for my patient? If I know there is a chance their own GP will not refer, is it not my duty to refer?

There is no doubt that C2C referrals are increasing but we have no evidence that this is being done for spurious reasons, in the main, and very good evidence that patients benefit and, at least, are not suffering because of it. The effect of peer reviews of primary care referrals and pressure from CCGs are certainly reducing referrals by primary care but , again, we have little evidence the reduction is due to the cutting away

of ‘inappropriate ‘ referrals and it may well be C2Cs are picking up the slack.

Before too much energy is put into such schemes, I would ask that we look at the reasons behind such referrals and also whether such referral management is being done for the patients’ sake or for other reasons.

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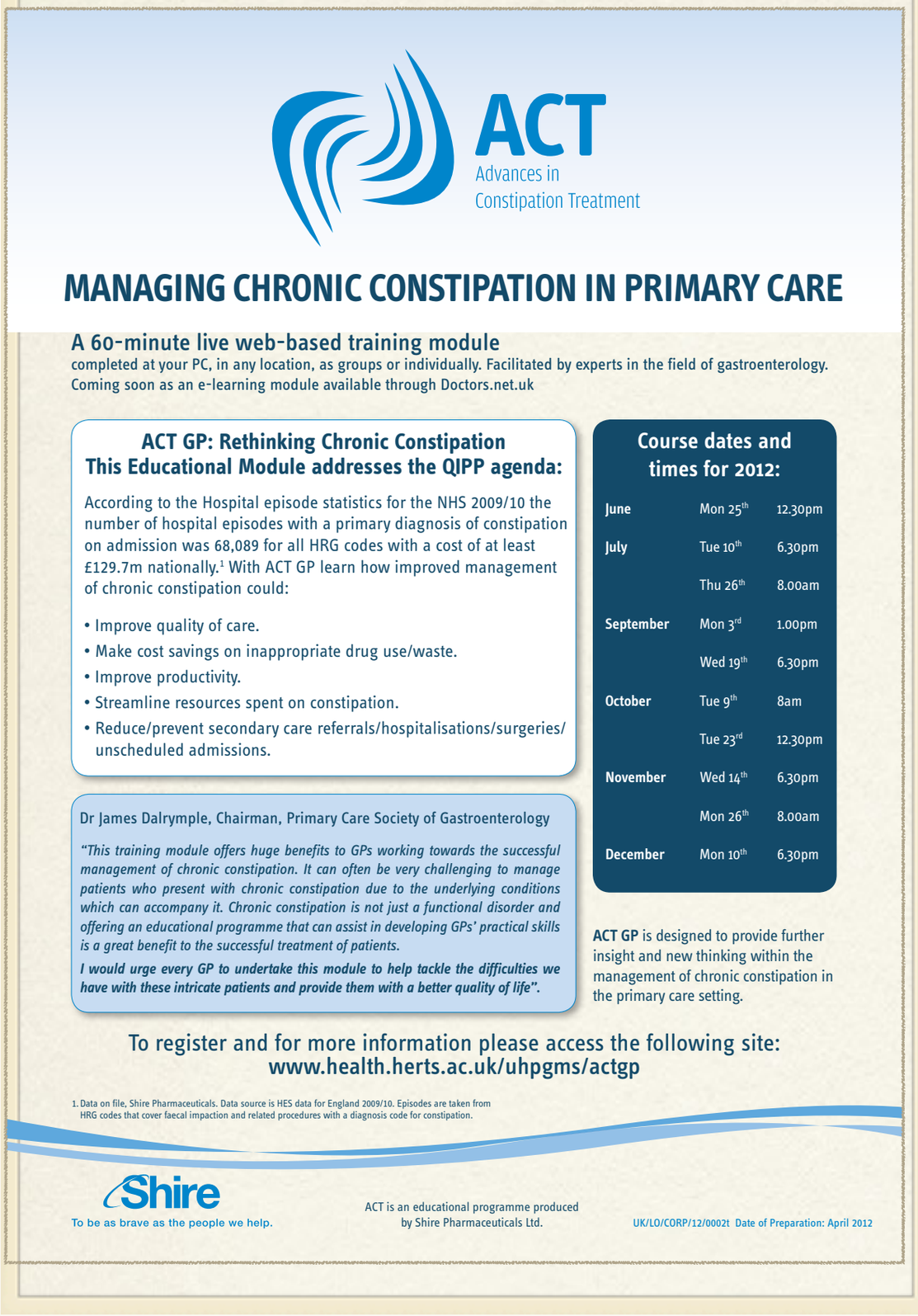

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