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GastroIntestinal Endoscopy

newsletter the insider

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Our Mission at GastroIntestinal Endoscopy is to deliver experienced and accessible endoscopy services with the highest quality of healthcare standards to improve the health outcomes of patients and the communities we serve.

GIE operates an Open Access Endoscopy Service from four locations:

- **SUNNYBANK** – Brisbane Endoscopy Services
- **CHERMSIDE** – Chermside Day Hospital
- **EVERTON PARK** – North West Private Hospital
- **AUCHENFLOWER** – The Wesley Hospital

Dr Hugh Spalding

Non-celiac Gluten Sensitivity: Is it real?

Gastrointestinal upset related to wheat ingestion in non-celiac patients is a world-wide problem. Thirty percent of a large Italian cohort of irritable bowel syndrome (IBS) patients were found to be wheat sensitive (somewhat of a problem in the home of pasta). The concept of non-celiac gluten sensitivity has been around for more than 30 years, but only recently has well conducted research begun. The presentation of non-celiac gluten sensitivity (NCGS) can include, abdominal pain bloating, constipation, diarrhoea, fatigue, headache, rash/eczema, aches and pains and paraesthesia.

Unfortunately the nomenclature for wheat related disorders has been confused which hasn't helped our understanding of them. A multidisciplinary task-force has addressed this recently and recommended gluten related disorders as the umbrella term which includes celiac disease and non-celiac gluten sensitivity for patients in whom coeliac disease has been excluded (Ludvigsson et al. 2013). Wheat allergy comprises a group of IgE mediated (and possibly some non-IgE mediated) disorders involving gluten and other wheat allergens. There remains controversy about the use of the term "non-celiac wheat sensitivity" which would encompass many patients with intolerance to short chain carbohydrates (FODMAPs) and is not part of the Oslo definitions. Wheat can also play a role in eosinophilic gastrointestinal disorders such as eosinophilic oesophagitis.

Although there are no biomarkers for NCGS a variety of laboratory evidence has been used to support its existence. NCGS has been associated with IgG antigliadin antibodies in 25–56% of cases (<8% in normal population) but not with the more specific celiac antibodies including IgG deamidated gliadin peptide antibody, IgA tTGA and IgA endomysial antibodies. Some have found a slightly higher prevalence in NCGS of HLA-DQ2 and DQ8 genotypes (50%) than normal (but others have found no correlation with these genotypes). Activation of circulating basophils and eosinophil and lymphocyte infiltration of the intestinal wall have also been described. Whilst there may be some evidence for a role of the innate immune response in gluten sensitivity, there is no conclusive evidence of activation of the adaptive immune response (including no increase in deamidated gliadin IgG). Unfortunately the interpretation of these findings is difficult, in part due to variable inclusion criteria for NCGS in different studies.

In addition many studies haven't adequately addressed the potential for non-gluten components of wheat to trigger symptoms (e.g. carbohydrate or other proteins). A recent trial in Melbourne involved patients carefully selected for IBS without CD who were given a gluten free (but otherwise uncontrolled) diet then randomised to a diet with or without gluten. Patients given gluten had higher levels of abdominal pain, bloating and tiredness and dissatisfaction with stool consistency. However, a subsequent study by the same group found a consistent reduction in symptoms with a low FODMAP diet but failed to find an increase after gluten challenge when on the low FODMAP diet. They concluded that "Non-celiac

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Image source: Wikipedia

Non-celiac Gluten Sensitivity: Is it real?

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gluten sensitivity is an entity awaiting validation, better diagnostic criteria, and, if it does exist, pathogenic mechanisms" (Biesiekierski et al 2013).

What to make of this? NCGS has generated significant interest in the general public and provision of gluten free food is big business. A Google search of this term returned about 262,000 results vs 456 in PubMed and the gluten free market is expected to be around $1.7 billion annually in the USA by 2015. The problem is not going away. Most patients who believe they are gluten sensitive have not been adequately assessed and in a recent prevalence study in the population is not recommended. Screening for Barrett's oesophagus is however suggested in patients with multiple risk factors associated with oesophageal carcinoma (age greater than 50, male, Caucasian, chronic gastro-oesophageal reflux, elevated body mass index).

Once Barrett's oesophagus has been identified, patients are entered into endoscopic surveillance programmes. This strategy is based on the presumption of progression from non-dysplastic Barrett's oesophagus through low-grade dysplasia to high-grade dysplasia and cancer. Recent studies have suggested the rate of progression of non-dysplastic Barrett's to cancer is 0.1% per patient year while the rate of progession from low-grade dysplasia to cancer is 0.5% per patient year and the rate of progression from high-grade dysplasia to cancer is 5–8% per year.

Surveillance endoscopy involves careful inspection of the region of Barrett's oesophagus. Traditionally endoscopists have relied on random biopsy sampling techniques to identify dysplasia, four quadrant biopsies every 1–2cms of Barrett's oesophagus, preferably using jumbo biopsy forceps. This technique does however have significant biopsy sampling error and with the recent availability of high resolution white light endoscopy, narrow band imaging and chromoendoscopy it has been appreciated that dysplasia is associated with visible abnormalities in most

Dr William Robinson

Oesophageal Perspectives *Part 1*

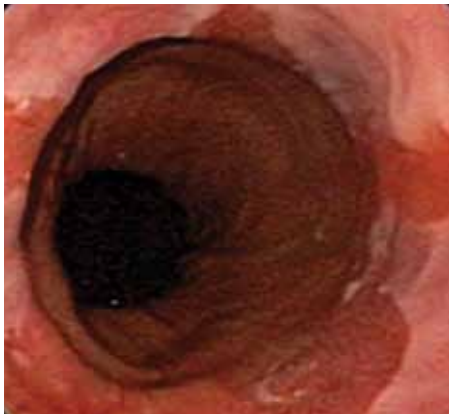

Image source: Up To Date

BARRETT'S OESOPHAGUS

Barrett's oesophagus is defined as "the condition in which metaplastic columnar epithelium that predisposes to cancer development, replaces the stratified squamous epithelium that normally lines the oesophagus". The metaplastic epithelium develops as a result of chronic gastro-oesopahgeal reflux disease. Barrett's oesophagus is of clinical importance because it is the only known precursor of oesophageal adenocarcinoma. The incidence of this malignancy has increased dramatically over the past four decades and over 75% of oesophageal cancers now detected are adenocarcinoma. The 5 year survival rate remains below 20% but survival is better when the cancer is detected early. This provides the rationale for endoscopic and histologic screening of Barrett's oesophagus to detect dysplastic change and early cancer.

Barrett's oesophagus is more common in men than women (ratio of 3:1) and the prevalence plateaus around age 50. Up to 10% of patients with gastro-oesophageal reflux disease will have Barrett's oesophagus at endoscopy and the likelihood of Barrett's mucosa increases with longer duration of gastro-oesophageal reflux related symptoms. Barrett's oesophagus is also present in approximately 2.6% of asymptomatic adults. From a population perspective then, about 40% of patients with Barrett's oesophagus are asymptomatic. This creates a dilemma for effective surveillance as any screening programme that relies on symptoms will not affect 40% of those at risk. Only 5% of oesophageal adenocarcinoma are currently discovered in patients with previously diagnosed Barrett's oesophagus.

The recommendation that patients with Barrett's oesophagus undergo endoscopic surveillance is based upon the assumption that Barrett's oesophagus adversely influences survival and surveillance can reduce mortality. There is however, no evidence demonstrating that population screening for Barrett's oesophagus leads to an improvement in survival or decrease in cancer related death and therefore screening in the general

USA only about 9% of people on a GFD actually had celiac disease.

In practical terms it is important to diagnose celiac disease in these people to avoid the long-term consequences of uncontrolled disease. Conversely those without coeliac disease can have a more liberal symptom-directed diet and family screening can be avoided. Several algorithms have been developed but these tend to ignore FODMAP intolerance.

In short:

- Celiac disease is differentiated with the use of serology and small bowel biopsies on gluten containing diet, sometimes with the assistance of HLA-DQ2 and DQ8 genotyping.
- Wheat allergy (typically IgE mediated disease) diagnosis involves skin prick tests, wheat specific IgE assays and the involvement of an allergist.
- The diagnosis of NCGS is one of exclusion and the input of a skilled dietician may be helpful to exclude FODMAP intolerance.

Biesiekierski J.R., Muir J. G. & Gibson P.R. *Is gluten a cause of gastrointestinal symptoms in people without celiac disease? Current Allergy and Asthma Reports* (2013) 13: 631–638

Ludvigsson J.F. , Leffler D.A, Bai J.C. et al *The Oslo definitions for coeliac disease and related terms. Gut* (2013) 62: 43–52

cases. Surface application of acetic acid with enhanced magnification is useful for improved visualisation of Barrett's mucosa, and this technique is used in our practice. The emphasis of tissue sampling of Barrett's oesophageal mucosa is therefore shifting towards targeted biopsies, in addition to random sampling.

Although current data suggests that the risk of cancer for patients with short segment Barrett's oesophagus is less than for patients with long segment disease, the two are managed in a similar manner. The timing of endoscopic surveillance for patients with Barrett's oesophagus is as follows:

- No dysplasia – 3–5 years
- Low-grade dysplasia – 6–12 months
- High-grade dysplasia – eradication therapy is recommended.

Management of gastro-oesophageal reflux disease in patients with Barrett's oesophagus involves similar principles to the treatment of the patients who have GERD without Barrett's oesophagus. The primary goal of anti-reflux therapy for these patients is to control reflux symptoms. Available data suggests that aggressive anti-reflux therapy, either pharmacologic or surgical, may reduce the likelihood of progression to high grade dysplasia or adenocarcinoma in patients with Barrett's oesophagus.

GP Support Needed for NBCSP

The Cancer Council has urged GPs to support the National Bowel Cancer Screening Program (NBCSP). Cancer Council Australia CEO, Professor Ian Olver, said GP support was key to foster participation, which is currently around 35%.

"Screening for bowel cancer with faecul occult blood test (FOBT) is one of the most clinically and economically effective public health measures available to Australians," Professor Olver said.

"While the screening program is based on participants taking their FOBT at home, GPs nonetheless have a critical role – in referring patients who test positive, in assisting with follow-up and in encouraging patients to take the test in the first place."

The campaign follows the announcement in the budget that $95.9 million will be allocated over four years to implement biennial screening for all Australians aged 50–74 between 2015 and 2020.

Currently, Australians aged 50, 55, 60 and 65 are eligible for the screening program. People aged 70 and 74 will be invited to participate next year.

Frequently Asked Questions Dr Neville Sandford

Q What tests should be performed for investigation of a positive FOBT?

A There are two major types of Faecal Occult Blood Tests (FOBTs), chemical tests (gFOBTs)which detect faecal haem which perioxidises the reagent guaiac, and immunological tests (FITs)which detect faecal globin. As blood passes through the gastrointestinal tract the globin is digested so FITs are negative during upper gastrointestinal bleeding, but gFOBTs may remain positive. Chemical tests are two to three times less sensitive than immunological tests for detecting blood in the stool, and also less specific as other peroxidases in the diet may cause false positive gFOBTs. Chemical tests also require sampling from at least three consecutive stools to improve their sensitivity whereas FITs are sensitive with just two stool samples. For these reasons the National Bowel Screening Programme (NBCSP) uses the immunological test as the FOBT of choice. Guaiac based tests are still used in the Australian Rotary Health programme and in Bowelscan testing.

If a patient has a positive FIT as a participant in the NBCSP, the recommended test is a colonoscopy. An upper endoscopy is not necessary as this test does not detect bleeding from the UGIT.

Q How often are FOBTs positive in the NBCSP?

A About one in 14 patients (7%) have a positive FOBT and most of these patients require further investigation as they are 12 times more likely to have colorectal cancer than patients with negative tests. In FOBT positive patients, 6% are found to have bowel cancer, 14% advanced adenomas, and 35% polyps. It is important to realise that a negative FOBT does not exclude cancer (it only detects blood, if present, in the stool, not polyps or cancer), so a symptomatic patient should be referred for colonoscopy without having a FOBT performed as a negative test may lull the patient or doctor into a false sense of security. Similarly it is recommended that patients already enrolled in surveillance programmes for follow-up of colonic polyps should not have regular FOBTs as a means of deciding whether to proceed with their follow-up investigation or not.

If you require A5 referral pads, please contact one of our four locations below. Electronic referral templates can be downloaded from our website www.gastros.com.au

GIE practice locations and contact details For all appointments, call 1300 4 GASTRO (1300 4 427876)

Brisbane Endoscopy Services

Suites 16–18
McCullough Centre
259 McCullough Street
Sunnybank QLD 4109

Phone: 07 3344 1844
Fax: 07 3344 2739

Chermside Day Hospital

Level 1
Chermside Medical Complex
956 Gympie Road
Chermside QLD 4032

Phone: 07 3120 3407
Fax: 07 3120 3443

The Wesley Hospital

3rd Floor, East Wing
451 Coronation Drive
Auchenflower
QLD 4066

Phone: 07 3870 3799
Fax: 07 3870 5069

North West Private Hospital

Endoscopy Unit
137 Flockton Street
Everton Park
QLD 4053

Phone: 07 3353 3322
Fax: 07 3353 9325

Private practice locations and contact details

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