

20th United European Gastroenterology Week 2012 (UEGW12)

Conference Review

Making Education Easy

UEGW, 20–24 October, 2012, Amsterdam, The Netherlands



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ABBREVIATIONS USED:

CsA	= cyclosporin
EO	= eosinophilic oesophagitis
EUS	= endoscopic ultrasound
MDR	= multidrug-resistant
UC	= ulcerative colitis
VA	= villous atrophy

About Research Review

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Welcome

to the United European Gastroenterology Week 2012 (UEGW12), the 20th Anniversary Congress of the United European Gastroenterology Federation, which was held in Amsterdam from October 20–24, 2012. This Conference Review is a locally focused summary of some of the latest advances in clinical management and cutting-edge basic and translational research in gastroenterology and hepatology presented at UEGW12.

The UEGW has been organised annually since 1992 and has become the largest and most prestigious GI meeting in Europe, attracting over 14,000 physicians, researchers and academics from around the world.

This Review has been created to allow those unable to attend, but with a keen professional interest in gastroenterology and hepatology research, to access a summary of significant clinical studies presented that are likely to affect current practice. Selection and review of the research has been carried out independently by David Rowbotham (Gastroenterologist, Hepatologist and Endoscopist, Auckland) and Alasdair Patrick (Consultant Gastroenterologist and General Physician, Auckland), who attended UEGW12.

I hope you find the Conference Review stimulating and I look forward to your feedback.

Kind regards

Chris Tofield

christofield@researchreview.co.nz

Oral versus intravenous esomeprazole in the treatment of acute peptic ulcer bleeding

Authors: Sung JJ et al

Summary: Outcomes are reported from an investigation into the efficacy of oral versus intravenous (IV) esomeprazole in the prevention of recurrent bleeding from peptic ulcer disease. A total of 224 patients with Forrest I or IIa/b peptic ulcer bleeding were initially treated with combination endoscopic therapy (adrenaline [epinephrine] injection combined with thermal coagulation) to control active bleeding, prior to randomisation to oral esomeprazole (40 mg twice daily for 3 days; n=115) or IV esomeprazole (80 mg IV bolus followed by 8 mg/h for 72 hours; n=109) after endoscopy. All patients received oral esomeprazole 40 mg/day after Day3 until repeated endoscopy at 8 weeks. Demographics and characteristics were similar between the groups at baseline. Clinical recurrent bleeding was defined as haematemesis, melaena or drop of haemoglobin by >2 g/dL after transfusion and confirmed by endoscopic examination. No significant between-group differences were observed for recurrent bleeding rates within 72h or at 30 days and 56 days of follow-up. Similarly, 30-day mortality rates did not differ significantly between the groups.

Comment (DR): We know that intravenous (IV) omeprazole (80 mg bolus given before acute endoscopy and subsequent continuous infusion at 8 mg/hr for 72 hours) is effective at down staging high risk ulcers and reducing hospital stay in acute non-variceal upper gastrointestinal bleeding (UGIB), and high dose IV omeprazole reduces rates of rebleeding. We also know that oral PPI is effective. To date, however, all the trials of PPI in acute UGIB, whether IV or oral, have been placebo controlled and there have been no head to head comparative trials of IV versus oral formulations, so this study is a welcome addition. Preliminary results would suggest that oral esomeprazole is no less effective than IV esomeprazole in preventing rebleeding and mortality in the highest risk peptic ulcer lesions. Currently esomeprazole is not funded in NZ, but there are a number of alternative PPIs available. Although a class effect cannot always be presumed, this paper would suggest we could treat more of our patients with oral PPI therapy.

Reference: *Gut* 2012;61(Suppl. 3):A6. Abstract OP027.

<https://uegw.congress-online.com/guest/ID6256b0a50b0e1f/AbstractView?ABSID=842>

Proton pump inhibition improves dysphagia, endoscopic signs and oesophageal mucosal integrity in adults with eosinophilic oesophagitis

Authors: Van Rhijn BD et al

Summary: A cohort of 10 adult patients with oesophageal eosinophilia (>15 eosinophils per high-power field), predominant symptoms of dysphagia and/or food impaction and typical endoscopic signs of eosinophilic oesophagitis. Patients underwent upper endoscopy at baseline and after 8 weeks of esomeprazole 40 mg twice daily. At 8 weeks, dysphagia was significantly improved and endoscopic signs of eosinophilic oesophagitis showed improvement with a trend towards significance. Electrical tissue impedance spectroscopy measurements during endoscopy revealed significantly higher extracellular mucosal resistance after esomeprazole treatment compared to baseline values. Moreover, transepithelial electrical resistance measured in Ussing chambers tended to be higher after esomeprazole. Transmucosal flux of rhodamine (40kDa) dextrans in Ussing chambers decreased significantly after esomeprazole, whereas there was no such change in fluorescein flux.

Comment (DR): The prevalence of eosinophilic oesophagitis (EO) in NZ is unknown but we certainly see a number of cases in Auckland, usually presenting with recurrent dysphagia or acute oesophageal food bolus obstruction. Management options include dietary restriction and cumbersome interventions such as attempting to swallow metered doses of a fluticasone inhaler. My own personal experience, however, is that the majority of patients with EO do very well with total symptom resolution (or marked improvement) simply using standard dose oral PPI, and patients prefer the simplicity of this approach. Hence I read this abstract with great interest. Although describing data from only a very small number of patients, this practical treatment strategy seems worthy of further investigation and longer term follow up. Mind you, I have to point out I'm not a big fan of Ussing chambers and fluorescein flux ... whatever they are?

Reference: *Gut* 2012;61(Suppl. 3):A18. Abstract OP079.

<https://uegw.congress-online.com/guest/ID6256b0a50b0e1f/AbstractView?ABSID=892>

Duodenal bulb biopsies: are they a necessity in coeliac disease?

Authors: Kurien M et al

Summary: This prospective study evaluated the diagnostic yield of taking duodenal bulb biopsies in coeliac patients compared with controls. Of the 550 enrolled patients, all were undergoing clinically indicated oesophagogastroduodenoscopy: 153 had newly diagnosed coeliac disease and comprised Group 1 (CD: New Diagnosis), Group 2 consisted of 91 patients with established coeliac disease (CD: Remission) and Group 3 comprised 306 controls. A total of 9% of patients in Group 1 and 14% in Group 2 were more likely than controls to have villous atrophy in the bulb alone ($p < 0.0001$ for both comparisons). In a comparison of the histological lesion of the bulb with the distal duodenum, a discrepancy in the severity of the lesion between the two sites was found in 40% of patients in Group 1 and 23% in Group 2 compared with only 7% of controls ($p < 0.0001$ for both comparisons). A total of 24/35 patients in Group 1 and 28/36 in Group 2 had the more severe lesion in the bulb. One control was diagnosed with villous atrophy. This patient was HIV-positive with positive tissue transglutaminase (tTG) and negative endomysial antibodies (EMA); however, the HLA status was incompatible with coeliac disease.

Comment (DR): It is debated amongst Gastroenterologists whether duodenal biopsies for the exclusion or monitoring of coeliac disease should include just the second and/or third parts of the duodenum or whether a biopsy should also be taken from the duodenal bulb. Individual practice seems to vary between these two schools of thought. Traditionally it has been thought that bulbar Brunner's glands could cause difficulties with histological interpretation, but more recent studies have shown that the duodenal bulb may be the only site of the proximal duodenum to demonstrate actual villous atrophy. This prospective study suggests that a sizeable minority of patients with coeliac disease may be missed if biopsies are not taken specifically from the duodenal bulb. Time for everyone to suck it up and fall into line?

Reference: Gut 2012;61(Suppl. 3):A35. Abstract OP148.

<http://tinyurl.com/duodenal-bulb-biopsies>

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Disclaimer: This publication is not intended as a replacement for regular medical education but to assist in the process. The reviews are a summarised interpretation of the published study and reflect the opinion of the writer rather than those of the research group or scientific journal. It is suggested readers review the full trial data before forming a final conclusion on its merits.

Research Review publications are intended for NZ Medical Professionals

Rescue treatment with quadruple therapy (3-in-1 capsule of bismuth, metronidazole and tetracycline with omeprazole) for *H. pylori* eradication in patients with multidrug resistance to clarithromycin, fluoroquinolones and metronidazole

Authors: Muller N et al

Summary: This review of data from a named patient programme in France (ATU) sought to determine the efficacy of bismuth-based quadruple therapy with omeprazole, bismuth subcitrate potassium, metronidazole and tetracycline (OBMT) using a single-triple capsule of BMT in multidrug-resistant *H. pylori*. Ninety-seven adult patients were treated with 3 single Pylera® capsules (3-in-1 capsule containing bismuth subcitrate potassium 140 mg, metronidazole 125 mg and tetracycline 125 mg) once daily plus omeprazole 20 mg twice daily for 10 days. Eradication was confirmed by urea breath test at least 28 days after end of treatment. Seventy-eight patients (80%) reported eradication after treatment with OBMT (in 3 cases despite premature discontinuation of treatment).

Comment (DR): Antibiotic resistance in *H. pylori* is increasing around the world, including in NZ. Rates of clarithromycin and metronidazole resistance, in particular, seem to be becoming more commonplace. It has been argued, however, that sensitivity testing *in vitro* may not be so relevant *in vivo*. Nevertheless it is important to find novel ways to treat patients colonised by apparent resistant strains of *H. pylori*. This study reports impressively high (almost too impressive) successful eradication rates of *H. pylori*, even in patients with multiple previous eradication failures. Bismuth subcitrate is available in NZ as DeNol at selected pharmacies, but is not funded and so the patient has to bear the cost of these tablets, plus the tetracycline. So not cheap, but perhaps a small price to pay for successful eradication and a life free of *H. pylori*?

Reference: Gut 2012;61(Suppl. 3):A47. Abstract OP205.

<https://uegw.congress-online.com/quest/ID6256b0a50b0e1f/AbstractView?ABSID=1013>

Independent commentary by Dr David Rowbotham and Dr Alasdair Patrick



Dr David Rowbotham is Clinical Director and Consultant Gastroenterologist at Auckland City Hospital. He trained in Gastroenterology and Hepatology in the UK and came to NZ in 1999. Not known for possession of either a tie or a razor, David's appearance, dry wit and sarcasm draw inevitable comparisons with another tortured medical soul.



Dr Alasdair Patrick has been practising as a Gastroenterologist and General Physician at Middlemore Hospital in Auckland since 2007. He is a NZ trainee who did his fellowship in London at the Royal Free Hospital and then worked as a Consultant Gastroenterologist at Singapore General Hospital.



Patients with moderate to severe Crohn's disease maintained fistula closure with HUMIRA at Week 56²

33% patients in HUMIRA arm had complete fistula closure*

100% of HUMIRA patients with complete fistula closure at Week 26, continued to have complete fistula closure at Week 56²



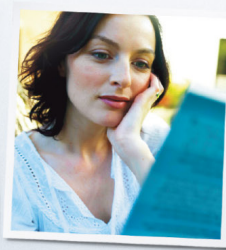
Before



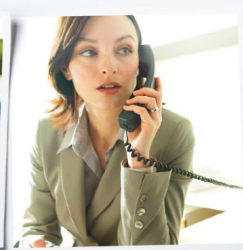
After



And after



And after



And after

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*Patients had luminal Crohn's disease with a CDAI >220 (CHARM study). Complete fistula closure defined as closure at the last two evaluations of all fistulas that were draining at baseline.

Reference 1: HUMIRA approved Data Sheet, V21. Reference 2: Colombel JF et al; CHARM study. Gastroenterology 2007;132:52-65 Humira Data Sheet. www.medsafe.govt.nz @ Registered Trademark. Abbott Laboratories NZ Ltd. 4 Pacific Rise, Mt Wellington. Before prescribing HUMIRA please review the Prescribing Information on page 4

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For more information, please go to <http://www.medsafe.govt.nz>

Infliximab vs oral cyclosporin in patients with severe ulcerative colitis refractory to intravenous steroids

Authors: Scimeca D et al

Summary: This study examined the efficacy and safety of infliximab versus oral microemulsion cyclosporin (CsA) in 30 patients with severe steroid-resistant ulcerative colitis (UC); 17 were randomly allocated to receive infliximab 5 mg/kg (induction regimen) and 13 to oral CsA 5 mg/kg. Responder patients at 1 month (Powell-Tuck index ≤ 3) started azathioprine 2 mg/kg/day. At baseline, demographics and characteristics did not differ significantly between the groups. At 1 month after randomisation, 9 infliximab-treated patients (53%) and 7 CsA recipients (54%) had achieved clinical remission ($p=0.96$). Over a mean 32-month follow-up, colectomy was performed in 7 patients treated with infliximab and 4 CsA-treated patients ($p=0.7$). The mean time for colectomy was 2.7 months (2 vs 4 in the infliximab and CsA groups, respectively; $p=0.2$). No serious adverse events were reported with CsA; 1 systemic cytomegalovirus infection and a death following pneumonitis were reported in the infliximab group ($p=0.5$). The crude mean cost of therapy was greater in the infliximab group (8,052.84€ vs 1,106.82€ for each patient, respectively).

Comment (DR): The management of acute severe (steroid refractory) ulcerative colitis has altered radically within the last few years with the use of both IV cyclosporin and induction regimen of infliximab as "rescue therapies". Both of these have been shown to be effective and to markedly reduce the need for colectomy. Published comparative studies, in general, show that there is not much to choose between them in terms of efficacy. Emerging results on the use of oral cyclosporin in this scenario (including our own data from Auckland City Hospital) show that it appears to be as effective as IV cyclosporin and a lot easier and cheaper to administer. This is the first trial I am aware of that directly compares the use of oral cyclosporin (albeit a lowish oral dose) with infliximab in acute severe ulcerative colitis and it would appear as though there is not much to choose between them other than significantly higher costs with infliximab. In little old cash-strapped NZ, this is welcome news indeed.

Reference: *Gut* 2012;61(Suppl. 3):A165. Poster Session P0292.
<http://tinyurl.com/b2Ue9nt>

Long-term follow-up of children with prenatal exposure to anti-TNF α therapy for maternal inflammatory bowel disease

Authors: Machkova N et al

Summary: These researchers assessed the impact of maternal exposure to anti-tumour necrosis factor alpha (anti-TNF α) agents on child development, in a cohort of 18 infants born to women with inflammatory bowel disease treated with anti-TNFs during pregnancy at a single centre. Sixteen children were exposed to infliximab and 2 to adalimumab during pregnancy with a median of 13 weeks between last application and birth. They were followed-up for a median 25 months. Fifteen children (83%) were born at term and 3 (17%) preterm with a median birth weight of 3050g. Except for mild jaundice in 4 children no perinatal complication was observed. One child had hip dysplasia; no congenital malformation was reported. All children had normal growth and neuropsychological development, except for one with suspected autism. Vaccination was given according to the national vaccination protocol in 16 infants (89%); vaccinations were postponed in 2 patients. One case of lymphadenopathy occurred after tuberculosis vaccination; no other adverse events were recorded. Of the 6 children diagnosed with atopic dermatitis, 4 had a positive parental history of atopy. Nine infants experienced ≥ 1 infectious complication, with the majority being respiratory. Eight children (44%) used ≥ 1 course of antibiotic treatment with 4 (22%) during the first year of life. Four children had measurement of infliximab from the cord blood with the levels ranging from 1.8 to 25 $\mu\text{g/mL}$. Immunological laboratory tests performed in 4 children revealed normal findings in all of them including serological response to vaccinations.

Comment (DR): Although Marla Dubinsky's data would suggest no specific harm to babies exposed to anti-TNF α agents in utero, concerns still remain amongst many NZ Gastroenterologists about the potential effects of prenatal exposure to biological agents. This observational study of a cohort of 18 children exposed to anti-TNF α agents during pregnancy showed no hint of any alarm signals compared to the general population. Of course a follow up period of only 2 years means that problematic issues could still emerge in later childhood, but there are no pointers at present to suggest this. So perhaps we can and should relax a little about this issue and reassure our pregnant (or planning-to-be-pregnant) patients that the real issue is how to keep their IBD under control and their pregnancies viable rather than be concerned about some unproven, hypothetical risks to baby that have not, to date, materialised.

Reference: *Gut* 2012;61(Suppl. 3):A280. Poster Session P0840.
<http://tinyurl.com/prenatal-anti-TNFa-exposure>

Incisional hernias and adhesion related complications – long term follow-up of a randomized trial comparing laparoscopic with open colon resection within a fast track program (Lafa study)

Authors: Bartels S et al

Summary: The Lafa study compared incisional hernia and adhesion-related small bowel obstruction (SBO) rates in the 2–5 years following randomisation to laparoscopic ($n=208$) or open colonic resection ($n=191$) for colon cancer within a fast track or standard care perioperative regimen. Median follow-up was 3.4 years. Significantly fewer incisional hernias were diagnosed in the laparoscopic group compared with the open group (21 [10.1%] vs 32 [16.8%]; $p=0.050$). In addition, significantly fewer patients had an episode of SBO in the laparoscopic group compared with the open group (5 [2.4%] vs 14 [7.3%]; $p=0.021$). After correcting for duration of follow-up in a Kaplan-Meier analysis, the reduction in incisional hernia rate was no longer significant ($p=0.069$), whereas the higher number of patients with an SBO episode did remain significant ($p=0.024$).

Comment (AP): This interesting follow-up from the Lafa study looks at what they call long-term follow-up of both the laparoscopic and the open groups and finds that at a median follow-up of only 3.4 years there was a 3-fold increased risk of small bowel obstruction in the open group. This is significantly greater than the laparoscopic group, which also has a higher rate of hernia that does not quite reach significance. That they found such a difference at an early stage surprises me and suggests that over time the benefits may be even greater.

This I think clearly shows that laparoscopic colectomy is the way the future is heading. We need to ensure that we encourage our surgical colleagues to offer this in NZ.

Abstract OP002.

<https://uegw.congress-online.com/quest/ID58ad3c0e120fd1/AbstractView?ABSID=653>

Appendicectomy as treatment for refractory left-sided ulcerative colitis

Authors: Pan AK et al

Summary: Outcomes are reported from 11 patients who underwent therapeutic appendicectomy for left-sided UC (median duration of symptoms 6 years) refractory to standard therapy consisting of 5-ASA ($n=11$), immunomodulators ($n=11$), CsA ($n=3$) and biologics ($n=9$). Patients were followed-up for a median 4 months. Appendiceal histology demonstrated ulcerative appendicitis ($n=1$), acute appendicitis ($n=1$), fibrous luminal obliteration ($n=5$) and normal ($n=4$). Following appendicectomy, mucosal appearances improved or completely healed in 6 patients. No improvement occurred in 2 patients and the endoscopic appearance deteriorated in 1 patient. Six patients have reported improvement in symptoms, with complete resolution in 4. One patient has required colectomy. Time to symptom improvement was on average 2.3 months (range from 1 to 10 months).

Comment (AP): This paper addresses a challenge we all face from time to time, as to what to do with refractory distal disease. Laparoscopic appendectomy is not a major procedure compared with the alternate and is a possible, albeit unproven option in this context. It was great to see some work from NZ presented in a plenary session and is a credit to those involved. The dramatic results seen in some cases raises the question of the role of the appendix in the condition. One wonders if comparing the appendix specimens from those who had a disease that responded and those who did not may yield clues as to the pathophysiology of the condition.

Abstract OP341.

<http://tinyurl.com/a7uml3g>

Diagnostic yield of EUS guided biopsy with 22F Pro-Core needles for upper gastrointestinal mass lesions: a randomised comparative study against FNA needles

Author: Nguyen NQ

Summary: The performance of two EUS-guided biopsy systems, 22F fine needle aspiration (FNA) and 22F Pro-Core (PC) needle, was compared in the evaluation of mass lesions within or adjacent to the upper gastrointestinal tract in 97 patients. Four needle passes were taken from each lesion and all specimens were prepared as cell-block for histocytological analysis. The PC group underwent biopsies for pancreatic ($n=40$), gallbladder ($n=2$), mediastinal ($n=3$) and oesophageal or gastric submucosal masses ($n=5$), while the FNA group underwent aspiration for pancreatic ($n=41$), mediastinal masses ($n=3$) and oesophageal or gastric submucosal lesions ($n=4$). Demographics and characteristics did not differ between the groups at baseline. Diagnostic yield was significantly higher with PC needles than with FNA needles (46/50 [92%] vs 36/47 [77%]; $p=0.03$). Core-like tissues were obtained from 32 patients (62%) in the PC group, allowing histological assessment and if required, further immunohistochemical evaluation.

Comment (AP): This paper compares standard FNA needle biopsy to a new needle that has a side cutting port. Core-like tissue is especially useful to determine if the lesion is a lymphoma. These samples can be obtained by the tru-cut and pro-core but not the FNA needle. Tru-cut needles are inflexible and nearly impossible to use in the duodenum due to their inherent stiffness and the angulation of the scope required. The study targeted all comers for needle biopsy and selected big lesions (2.4 and 2.6cm). They are not clear on the site in the pancreas of the lesions. Therefore for an experienced endoscopist (>3000 EUS cases) getting a core in only 62% of cases in big lesions (some that may have been distal) is not a great step forward. I would be interested in a study comparing the Tru-cut needle to Pro-core, especially in uncinata process lesions, as these are the ones we cannot get at easily with the available needles.

Poster P0198.

<http://tinyurl.com/baa4pd4>

Patients with endoscopically visible polypoid adenomatous lesions within the extent of ulcerative colitis have an increased risk of colorectal cancer despite endoscopic resection

Authors: Subramanian V et al

Summary: Long-term outcomes are reported for 301 patients with a confirmed histological diagnosis of UC from 1991–2004 and followed-up until June 2011. Twenty-nine of these patients had endoscopically visible adenomatous lesions removed by endoscopic resection. The crude incidence rate of developing colon cancer in patients with UC was 2.45 (95% CI 1.06 to 4.83) per 1000 person-years of disease (PYD) versus 11.07 (95% CI 3.59 to 25.83) per 1000 PYD in those with UC and polypoid adenomas within the extent of inflammation. The adjusted incidence rate ratio of developing colorectal cancer on follow-up in UC patients with polypoid dysplastic adenomatous lesions within the extent of inflammation was 4.35 (95% CI 1.40 to 13.45).

Comment (AP): Sporadic adenomas or adenoma-like masses in an area of inflammation can occur in UC. Evidence suggests that endoscopic treatment of these lesions can be performed but the risks of subsequent cancer are not clear. This study addresses the outcomes of finding and treating of adenoma lesions within an area of inflammation in UC. The adjusted incidence rate ratio in these patients is 4.35. We need to be aware of this increased risk and target our follow-up appropriately.

Poster P0325.

<http://tinyurl.com/Poster-P0325>

Helicobacter pylori eradication in East Malaysia: sequential vs standard 7 day triple therapy

Author: Singh M et al

Summary: This Malaysian study compared *H. pylori* eradication rates of a standard triple therapy regimen (rabeprazole 20 mg twice daily [bd], amoxicillin 1 g bd and clarithromycin 500 mg bd for 7 days) with those of a 10-day sequential therapy regimen (rabeprazole 20 mg bd and amoxicillin 1 g bd for the first 5 days; followed by rabeprazole 20 mg bd, clarithromycin 500 mg bd, metronidazole 400 mg three times daily for the next 5 days). All 100 study participants were *H. pylori*-positive with dyspepsia. Data were evaluable from 91 patients who underwent a UBT at 6 weeks after completing eradication therapy (42 received sequential therapy and 49 received standard triple therapy). On endoscopy, 57 patients had gastritis, 15 had erosions, 11 had Forrest III ulcers and 8 had normal endoscopic findings. The overall per-protocol eradication rate was 90.1%. Intention-to-treat eradication rates were 76% for sequential therapy and 88% for standard therapy. Between-group per-protocol eradication rates did not differ significantly (OR 0.926; 95% CI 0.232 to 3.699; $p=0.537$).

Comment (AP): There were a lot of posters on eradication rates for various regimens from countries including Iran, Korea, China, Portugal and Italy, to name a few. I have selected this abstract, as it did not find any significant difference between sequential and standard triple therapy similar to what we use in NZ. Evidence points to increasing resistance of this organism but exact treatment is not clear and will require local data. We await with interest the results from the Middlemore resistance study that will be presented at the NZSOG meeting later this month. I think we need to look at new guidelines for NZ.

Poster P0510.

<http://uegw.congress-online.com/guest/IDa8cdeb86d4120e/AbstractView?ABSID=1754>

Oesophageal dilations in eosinophilic oesophagitis

Authors: Ukleja A et al

Summary: Records were retrospectively reviewed from 22 patients (mean age 49 years) with eosinophilic esophagitis (EoE) who had undergone a total of 27 oesophageal dilations (through-the-scope balloon, $n=25$; Savary guidewire, $n=3$) at a single centre. Significant improvement was noted for 19 patients (86%) who underwent a single dilation. Two patients (9%) required 3 dilations and 1 patient (4.5%) required 2 dilations. Mean dilation size was 17.8 mm. The maximum size dilation used was 20 mm in 7 patients (21%), 19 mm in 3 patients (11%), 18 mm in 13 patients (48%) and 15 mm in 2 patients (7.5%). The rate of complications was very low (11%) and each of the 3 events were considered to be minor: very deep mucosal tear ($n=1$) and severe chest pain ($n=2$). No oesophageal perforations occurred. No patients required post-procedural hospitalisation. Eosinophil peak count was not associated with the complication rate.

Comment (AP): The risk of perforation from dilatation in this condition was thought to be high but recent meta-analysis has suggested that it may be as low as 0.1%. This series using TTS dilators from the Cleveland clinic again shows that the procedure is safe. All patients were steroid non-responders and most were dilated up to 18mm. No data on symptomatic outcomes were provided. They also looked at whether eosinophil count predicted risk of complication but the numbers are small. I wonder if it is the development of an increased fibrous mucosa that confers the risk of deep tears and not inflammation.

Poster P0435.

<https://uegw.congress-online.com/guest/IDf2a1070cc51010/AbstractView?ABSID=1681>

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PHARMAC Pharmaceutical Schedule: HUMIRA is fully subsidised under Special Authority for the treatment of adults with severe Crohn's disease. Refer to Pharmaceutical Schedule for full Criteria

PLEASE REVIEW FULL DATA SHEET BEFORE PRESCRIBING. The full Data Sheet is available on request from Abbott Laboratories NZ Ltd, 4 Pacific Rise, Mt Wellington, or by phoning 0800 73 72 71, or on the Medsafe website. Humira is a Prescription Medicine containing adalimumab 40 mg/0.8 mL for injection. **INDICATIONS: Crohn's disease (CD):** Treatment of moderate to severe CD in adults to reduce the signs and symptoms of the disease and to induce and maintain clinical remission in patients who have an inadequate response to conventional therapies, or who have lost response or are intolerant of infliximab. **CONTRAINDICATIONS:** Severe infections including sepsis, active TB, opportunistic; concurrent anakinra; moderate to severe heart failure. **PRECAUTIONS:** Infections (bacterial, mycobacterial, invasive fungal e.g. histoplasmosis, viral or other opportunistic); hepatitis B, latent TB; demyelinating disorders; haematologic events; live vaccines; immunosuppression; new or worsening CHF; renal, hepatic impairment; malignancy; hypersensitivity reactions; latex sensitivity; concurrent abatacept; elderly; pregnancy, lactation, surgery. **ADVERSE REACTIONS:** Respiratory tract infections, leucopenia, anaemia, headache, abdominal pain, nausea and vomiting, elevated liver enzymes, rash, musculoskeletal pain, injection site reaction are very commonly seen adverse events. Benign neoplasm and skin cancer including basal cell and squamous cell carcinoma were commonly reported. Fatal infections such as tuberculosis and invasive opportunistic infections have rarely been reported. For others, see full Data Sheet. **DOSAGE AND METHOD OF USE CD:** Induction 160mg sc (Four injections on Day 0 or Two injections on Day 0 and 1), 80mg as two sc injections on Day 14, then Maintenance: 40mg sc starting on Day 28 and continuing fortnightly. **DATE OF PREPARATION:** 28 January 2011 Version 12. TAPS PP9910. NZ-HUMG-2011-9. MW 41513.



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