RCPE: Gastroenterology & Hepatology Course 2022

Day 1: 3 February 2022

What is new in the management of gastroparesis? - Dr Ian Beales

Q. What about bypassing pylorus with gastro-jejunostomy?

A. Gastojejunostomy is definitely a surgical option for those patients with refractory gastroaparesis. However, it does seem that a surgical (laparoscopic) pyloromyotomy is probably the surgical treatment of choice. There are no randomized trial to inform this. A systematic review (*J Surg Res (*2018) 231:263-269) suggested that 55-65% of patients will gain significant benefit from pyloromyotomy in the cardinal symptoms of gastroparesis. A non-randomised by well-performed registry study (*J Gastroenterol Hepatol* (2021) 36: 3177-82) with 102 subjects did show that clinical success was higher for G-POEM than laparoscopic myotomy (92 vs 82%) and that complications were higher with surgery (21 versus 13%). The choice of intervention probably depends on expertise and availability but probably a gastrojejunostomy is not superior to a pyloromyotomy. A gastrojejunostomy may not be more effective because (1) the trituration effect of the gastric antrum is still missing (2) just improving gastric emptying by employing the effect of gravity does not clearly relate to symptom-improvement due to the other pathological mechanisms in play.

Q. When do we consider gastric pacemaker Vs surgery?

A. There are no comparative trials and this is probably partly related to availability. The two techniques are not mutually exclusive. It is important to remember that the electric stimulation is not actually a “pacemaker” and does not restore normal gastric myoelectrial activity (it is not akin to a cardiac pacemaker). The main benefit from the gastric electrical stimulation is in reduction in vomiting. There is very little evidence that the other symptoms are improved. Hence for those with most troublesome vomiting, electrical stimulation might be the best option. For other symptoms consider surgical pylomyotomy. However these are invasive procedures and should be reserved for only the most refractory patients and in most cases are not required,, if other medications and diet are optimised.

Q. Are patients with T2DM equally at risk vs T1DM?

A. The US data reported tat 50% of patients with gastroparesis had T2DM and 5 % had T1DM. The recent UK data reported that 19% of the gastroparesis patients had T1DM and 14% T2DM. The Mayo clinic (admitted very specialised reported that 71% of the diabetic patients in their cohort had T2DM. To put this in context, the prevalence of T1DM is about 0.2% and T2DM about 5%. So the absolute risk is higher for T1DM. It is important to note that in both T1 and T2 diabetes, gastroparesis can occur without any other diabetic complications (36% of T2 DM patients with gastroparesis)

Q. Do you use Botox in your practise?

A. Yes, I admit that I do use botox. After optimising diet and other drug medication, I do use botox. I appreciate that the 2 clinical trials had an overall negative effect. Although more recent data suggest a subset of patients, especially those with preserved antral motility may benefit more and improve. However due to the lack of availability of gastric manometry, we cannot phenotype these patients fully and hence this is an empirical treatment. Until this year, I have not had the ability to phenotype using the EndoFLIP. I use 200 units, in 4 x 50units injections around the pylorus. Botox in this context does seem to be safe and there are no real contradictions and certainly seems worth doing before more invasive surgical, endoscopic or nutritional interventions. Botox does seem to work in selected patients and certainly I have several that have required re-treatment at about 9 months, which seems to fit with the pharmacology of botox. It is of course impossible to unravel this from a placebo effect.

Q. Any drugs you would recommend to avoid GA gastropresis?

A. The two key messages regarding medications are (1) use any essential medications is a form that will be bioavailable, liquid formulations are usually absorbed normally in gastroparesis or alternatively use a non-oral route, such as the melts available for 5HT3 antagonsists and (2) avoid medications that clearly delay gastric emptying. As discussed the relationship between the absolute value of gastric emptying and symptoms is very controversial but it does seem prudent to avoid drugs known to delay gastric emptying or motility. The most important ones here are stopping smoking and avoiding opiates. At present there is no evidence that the available opiate antagonist drugs, such as naloxegol, can reverse the upper GI effects of opiates. The effect of tricyclic antidepressants is controversial and these can be continued in low doses but certainly high doses (> 50 mg daily) should be avoided. Against that the TCAs do not seem very effective for dyspeptic symptoms in those with delayed gastric emptying, although of course the are used for other indications. Calcium channel blockers such as diltiazem have not been convincingly shown to delay gastric emptying, although in the UK study, in about 5% of the whole gastoparesis cohort the cause was said to be calcium channel blockers. As there are few if any absolute indications for their use, it would be wise to use alternative agents. The use of cannabis should probably be avoided. Cannabis use is very prevalent in this population and constituents of cannabis definitely delay gastric emptying. Additionally cannabis use can be associated with nausea and vomiting as part of the cannabis hyperemesis syndrome. Of course nabilone has been used successfully as an amtiemetic in different contexts. On balance cannabis use should be advised against, but this emphasises the difficulty is using gastric emptying as a surrogate instead of overall symptom burden. It is probably not necessary to avoid antiemetics with anticholinergic effects such a cyclizine and diphenhydramine if these are helpful for the treatment of nausea and vomiting.