# Practical tips on Common Gastro Cases

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MMH

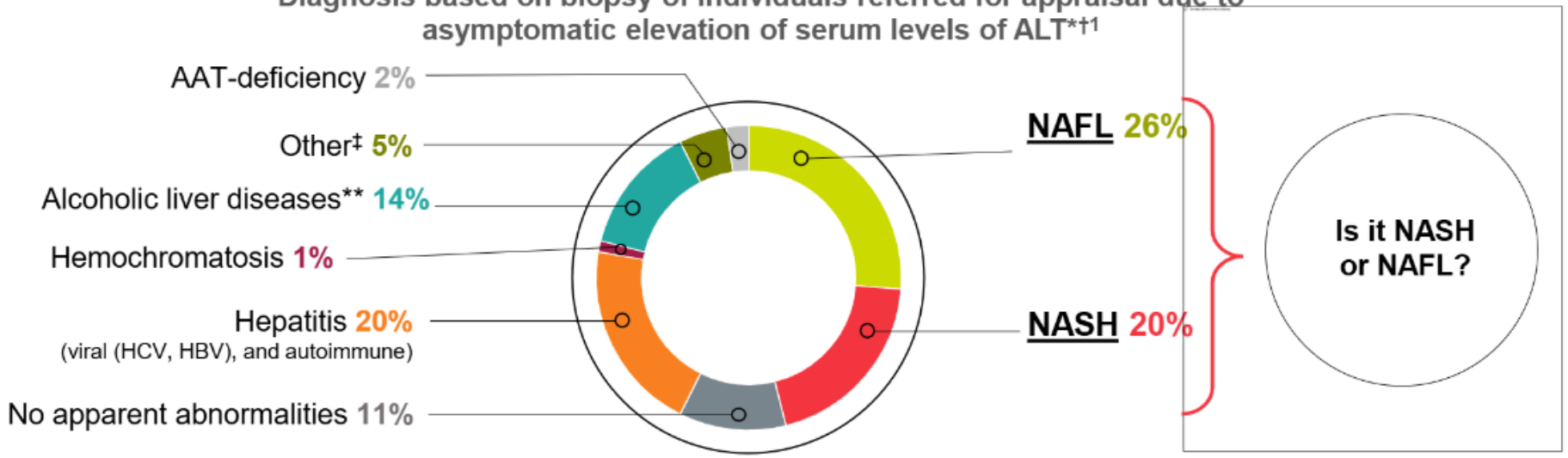
# Topics

- Abnormal liver function tests
- PPI use
- Polyp surveillance guidelines

# Elevated ALT: what are associated diagnoses?

## An example of the disease spectrum in Swedish patients referred for elevated ALT levels

Diagnosis based on biopsy of individuals referred for appraisal due to asymptomatic elevation of serum levels of ALT*†[1]




**After other liver diseases have been excluded, further investigation is needed to identify patients with NASH versus NAFL[2]**

*Based on liver biopsy of 256 Swedish individuals referred for appraisal because of asymptomatic elevation of serum levels of ALT. Inclusion criteria = persistently elevated levels of aspartate aminotransferase and alanine aminotransferase for longer than 6 months. For illustrative purposes only and not intended to inform management decisions. This population may not be representative of the US population.[1]
†List of liver diseases to exclude is not exhaustive; ‡Other defined as unspecific histopathological findings; **Alcoholic liver diseases: alcoholic steatohepatitis/alcoholic fatty liver disease = 10%; alcoholic liver disease = 4%.
AAT, alpha-1 antitrypsin; ALT, alanine aminotransferase; HBV, hepatitis B virus; HCV, hepatitis C virus; NAFL, nonalcoholic fatty liver; NASH, nonalcoholic steatohepatitis; US, United States.
1. Adapted from Soderberg C, et al. *Hepatology*. 2010;51:595–602; 2. Spengler EK, Loomba R. *Mayo Clin Proc*. 2015;90(9):1233–1246.

# Evaluation of abnormal liver tests

- Healthy ALT is <25-30 IU/mL in women, 30-35 IU/mL in men
- Non liver related causes:
- High ALP in pregnancy, vitamin D deficiency, hyperparathyroidism
- High AST in marathon runners

# Evaluation of abnormal LFTs

- Population based survey in US 1999 to 2002 estimated abnormal ALT in 8.9% of population using 45 Iu as cut off
- - 2015: 15% have high ALT if using <25 for women and <35 for men
- First step is to repeat the LFTs
- Many things can cause a one off elevation of liver tests
- Mild elevations should be monitored for 6 months before full work up
- Good history: alcohol, medications
- Hepatitis serology
- General liver screen: ANA, tissue autoantibodies, immunoglobulins, alpha 1 antitrypsin, ceruloplasmin, ferritin

# Case 1- TP

- 43 year old Maori man seen in clinic since 2015
- Chronic hepatitis B, eAg negative
- ALT 269
- HBV DNA 7 log IU/mL
- Started entecavir in 2015
- Shear wave elastography 2016 cirrhosis
- History of epilepsy on lamotrigine

## Hepatitis B DNA Quantitation

| | 01/06/16 13:30 | 24/10/18 12:30 | 01/02/19 13:49 | 12/08/21 14:17 | 18/02/22 13:47 | 20/09/22 11:09 | 20/01/23 12:57 | 20/10/23 08:58 | 09/04/24 08:52 |
|---|---|---|---|---|---|---|---|---|---|
| Specimen type | | | | | | | | | Serum |
| HBV DNA Viral load | 61202312 | 9455984 | 7435 | 26500000 | 404 | 44300000 | 2140000 | 7350 | 853 |
| HBV DNA | 7.79 | 6.98 | 3.87 | 7.42 | 2.61 | 7.65 | 6.33 | 3.87 | 2.93 |
| LabPlus Reference Number | | | | | | | | | |
| Result From | | | | | | | | | |
| Specimen type | Plasma | Serum | Serum | Serum | Serum | Serum | Serum | Serum | |

LabPlus Reference Number 09/04/24 08:52
17WN00862800

Result From 09/04/24 08:52
LabPlus Auckland

HBV DNA 18/02/22 13:47
As of 05/07/2021 this test is now performed on the Cobas 6800 system (Roche Diagnostics).

HBV DNA 12/08/21 14:17
As of 05/07/2021 this test is now performed on the Cobas 6800 system (Roche Diagnostics).

HBV DNA 01/06/16 13:30
This result was determined using the Roche COBAS/Taqman HBV version 2 test, which has a linear range of 20 HBV DNA IU/mL to 1.7E+8 HBV DNA IU/mL. (One HBV DNA IU is equivalent to 5.82 HBV DNA copies.)
Note: Results are expressed both as the number of HBV DNA IU/mL, also as the logarithm of the number of HBV DNA IU/mL.

| | 27/06/20 09:11 | 07/09/20 13:39 | 09/09/20 11:02 | 14/01/21 12:31 | 23/03/21 12:04 | 13/04/21 13:35 | 12/08/21 14:17 | 19/01/22 12:09 | 18/02/22 13:47 |
|---|---|---|---|---|---|---|---|---|---|
| Bilirubin | 5 | 18 | 24 | 5 | 4 | 5 | **32** | 13 | 6 |
| Alkaline phosphatase | 105 | **137** | 105 | 110 | 84 | 93 | **142** | 84 | 107 |
| GGT | **133** | **244** | **280** | **130** | **74** | **85** | **131** | 42 | 33 |
| ALT | 33 | **243** | **157** | 25 | 16 | 14 | **323** | 23 | 15 |
| Protein | 68 | 72 | 79 | 69 | 73 | 79 | 70 | 77 | 73 |

Sep 2020- ALT flared from normal to 243. What should we do now?

# Question 1- What should we do first?

- 1) Change antiviral treatment
- 2) Check compliance
- 3) Look for alternative causes of abnormal liver tests
- 4) USS

# Question 2: What do we do next?

- 1) Change antiviral treatment
- 2) Check compliance
- 3) Look for alternative causes of abnormal liver tests
- 4) USS

# March 2024

| | 20/10/23 08:56 | 20/10/23 08:58 | 07/03/24 12:33 | 07/03/24 12:34 |
|---|---|---|---|---|
| Bilirubin | 19 | 18 | 160 | 158 |
| Alkaline phosphatase | 93 | 92 | 115 | 114 |
| GGT | 47 | 45 | 85 | 84 |
| ALT | 36 | 39 | 287 | 289 |

- INR 1.0, Albumin normal
- AFP 93
- No encephalopathy or ascites

# March 2024

- Reports compliance with tablets “never misses”
- Currently on:
- Tenofovir 245mg daily
- Loratadine 10mg daily
- Lamotrigine 400mg bd
- Carbamazepine 300mg daily

# Question 3: What do you think is happening now?

- 1) Flare of hepatitis B
- 2) Antiviral resistance
- 3) HCC
- 4) Biliary obstruction
- 5) Decompensated liver disease
- 6) Drug induced liver injury
- 7) Alcoholic hepatitis

# Question 4:  What would you do next?

- 1) Check antiviral resistance
- 2) Imaging
- 3)  Check drug levels

- Triphasic CT liver no HCC
- HBV DNA 4.7 log IU/mL
- Carbamazepine levels therapeutic
- Lamotrigine levels elevated 23 (3-15)
- DILI suspected- has been on lamotrigine for a long time but on quite high doses
- Carbamazepine started <6 months ago
- Carbamazepine stopped after neurology review
- Lamotrigine dose reduced (as carbamazepine induces metabolism of Lamotrigine)

**HBV drug resist** *Accepted by Tien Huey Lim (CMD)*

HBV drug resistance
Genotypic sensitivity predictions are based on GRADE's HBV-Resistance interpretation tool algorithm version 07-2019 {https://www.hiv-grade.de/}

| Anti Viral Drug | | Mutation |
|---|---|---|
| 3TC | Lamivudine | S |
| ADV | Adefovir | S |
| ETV | Entecavir | S |
| LdT | Telbivudine | S |
| TDF | Tenofovir | S |

HBV drug resistance
Genotype and percent similarity to closest reference isolate
D(96.5%)

Resistance - R {High level resistance}
S {Susceptible}
I {Intermediate level resistance}

HBV drug resistance comment

| | 08/03/24 17:53 | 09/03/24 09:19 | 12/03/24 08:56 | 09/04/24 08:52 | 28/05/24 11:56 |
|---|---|---|---|---|---|
| Bilirubin | ✦163 | ✦153 | ✦121 | 24 | 17 |
| Alkaline phosphatase | ✦111 | ✦108 | ✦105 | 112 | 81 |
| GGT | ✦91 | ✦89 | ✦87 | 48 | 45 |
| ALT | ✦242 | ✦215 | ✦143 | 22 | 20 |

# Carbamazepine induced liver toxicity

- Benign elevation of ALT and GGT in 1-22% of patients
- 16/10,000 clinically significant hepatotoxicity
- Time course usually within 6 months (median 5 weeks) after treatment initiation
- Can be few years after
- Resolution of jaundice occurs within 5-7 days of stopping the drug

# Drug induced liver injury

- Any drug can cause any liver injury!!
- Higher risks in women, older age, chronic liver disease, higher dose
- Can be at start of medication, or in some cases “any time” during therapy or after stopping therapy
- Should rule out other causes of liver injury

# Drug Induced Liver Injury. DILI, SILI, MILI, HILI

| Hepatocellular (Elevated ALT) | Mixed (Elevated ALP + Elevated ALT) | Cholestatic (Elevated ALP + TBL) |
|---|---|---|
| Acarbose | Amitriptyline | Amoxicillin–clavulanic acid |
| Acetaminophen | Azathioprine | Anabolic steroids |
| Allopurinol | Captopril | Chlorpromazine |
| Amiodarone | Carbamazepine | Clopidogrel |
| Baclofen | Clindamycin | Oral contraceptives |
| Buproprion | Cyproheptadine | Erythromycins |
| Fluoxetine | Enalapril | Estrogens |
| HAART drugs | Flutamide | Irbesartan |
| Herbals: kava kava and germander | Nitrofurantoin | Mirtazapine |
| Isoniazid | Phenobarbital | Phenothiazines |
| Ketoconazole | Phenytoin | Terbinafine |
| Lisinopril | Sulfonamides | Tricyclics |
| Losartan | Trazodone | |
| Methotrexate | Trimethoprim–sulfamethoxazole | |
| NSAIDs | Verapamil | |
| Omeprazole | | |
| Paroxetine | | |
| Pyrazinamide | | |
| Rifampin | | |
| Risperidone | | |
| Sertraline | | |
| Statins | | |
| Tetracyclines | | |
| Trazodone | | |
| Trovafloxacin | | |
| Valproic acid | | |

**Table 3.** Medications, Herbs, and Drugs or Substances of Abuse Reported to Cause Elevations in Liver-Enzyme Levels.

**Medications**

Antibiotics
- Synthetic penicillins
- Ciprofloxacin
- Nitrofurantoin
- Ketoconazole and fluconazole
- Isoniazid

Antiepileptic drugs
- Phenytoin
- Carbamazepine

Inhibitors of hydroxymethylglutaryl–coenzyme A reductase
- Simvastatin
- Pravastatin
- Lovastatin
- Atorvastatin

Nonsteroidal antiinflammatory drugs

Sulfonylureas for hyperglycemia
- Glipizide

**Herbs and homeopathic treatments**

Chaparral

Chinese herbs
- Ji bu huan
- Ephedra (mahuang)

Gentian

Germander

Alchemilla (lady's mantle)

Senna

Shark cartilage

Scutellaria (skullcap)

**Drugs and substances of abuse**

Anabolic steroids

Cocaine

5-Methoxy-3,4-methylenedioxymethamphetamine (MDMA, "ecstasy")

Phencyclidine ("angel dust")

Glues and solvents
- Glues containing toluene
- Trichloroethylene, chloroform

# DILI

- Paracetamol is the most common cause of DILI in USA, also the most common cause of acute liver failure
- Normal dose for paracetamol toxicity is 6-12g/day, but toxicity can occur at much lower doses in certain circumstances
- - Alcohol use
- Fasting state

# DILI

- High index of suspicion
- Review all medications
- Check time course of medications (although can occur at any time)
- Check OTC meds or herbal meds

# Case 2- MC

- 72 year old Chinese female presented with jaundice and abnormal LFTs 20/1/23
- 1) Chronic gastritis, recent gastroscopy 14/11/22, gastric stenosis at pylorus- on omeprazole
- 2) Bronchiectasis
- 3) post nasal drip/sinusitis

# Case 2 MC

Previous results

| | Awanui Labs | CMDHB | | CMDHB | Awanui Labs | Awanui Labs |
|---|---|---|---|---|---|---|
| | 11/01/22 16:30 | 12/01/22 15:12 | | 24/03/22 10:33 | 13/10/22 11:41 | 19/01/23 14:20 |
| Bilirubin | 9 | | | | 19 | **296** |
| Alkaline phosphatase | 66 | | | | 81 | **221** |
| GGT | 18 | | | | **76** | **210** |
| ALT | 18 | | | | **63** | **477** |

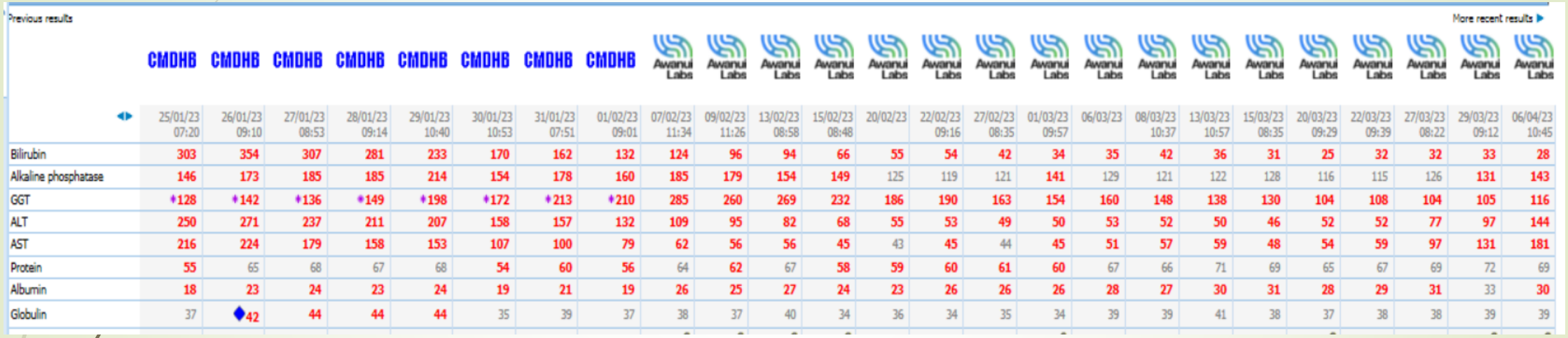
# Case 2- MC

- INR 1.5
- USS small volume ascites, mildly nodular right lobe of liver
- AFP 23
- CMV/EBV negative
- Hepatitis serology negative
- Liver autoantibodies negative
- Immunoglobulins IgG 22.4, IgA 4.4, IgM 2.6
- Ceruloplasmin/alpha 1 antitrypsin negative
- ANA 1:1260, dsDNA 160, F-Actin antibodies positive

# Question 5: what would you do next?

- 1) Liver biopsy
- 2) Triphasic CT liver
- 3) Empiric trial of steroids

- Liver biopsy : plasma cells, interface hepatitis, cholestasis
- Started on steroid treatment for presumed autoimmune hepatitis
- ?DILI due to omeprazole (rare)
- Omeprazole stopped and changed to famotidine



Previous results — More recent results ▶

| | CMDHB | CMDHB | CMDHB | CMDHB | CMDHB | CMDHB | CMDHB | CMDHB | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs | Awanui Labs |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 25/01/23 07:20 | 26/01/23 09:10 | 27/01/23 08:53 | 28/01/23 09:14 | 29/01/23 10:40 | 30/01/23 10:53 | 31/01/23 07:51 | 01/02/23 09:01 | 07/02/23 11:34 | 09/02/23 11:26 | 13/02/23 08:58 | 15/02/23 08:48 | 20/02/23 | 22/02/23 09:16 | 27/02/23 08:35 | 01/03/23 09:57 | 06/03/23 | 08/03/23 10:37 | 13/03/23 10:57 | 15/03/23 08:35 | 20/03/23 09:29 | 22/03/23 09:39 | 27/03/23 08:22 | 29/03/23 09:12 | 06/04/23 10:45 |
| Bilirubin | 303 | 354 | 307 | 281 | 233 | 170 | 162 | 132 | 124 | 96 | 94 | 66 | 55 | 54 | 42 | 34 | 35 | 42 | 36 | 31 | 25 | 32 | 32 | 33 | 28 |
| Alkaline phosphatase | 146 | 173 | 185 | 185 | 214 | 154 | 178 | 160 | 185 | 179 | 154 | 149 | 125 | 119 | 121 | 141 | 129 | 121 | 122 | 128 | 116 | 115 | 126 | 131 | 143 |
| GGT | ♦128 | ♦142 | ♦136 | ♦149 | ♦198 | ♦172 | ♦213 | ♦210 | 285 | 260 | 269 | 232 | 186 | 190 | 163 | 154 | 160 | 148 | 138 | 130 | 104 | 108 | 104 | 105 | 116 |
| ALT | 250 | 271 | 237 | 211 | 207 | 158 | 157 | 132 | 109 | 95 | 82 | 68 | 55 | 53 | 49 | 50 | 53 | 52 | 50 | 46 | 52 | 52 | 77 | 97 | 144 |
| AST | 216 | 224 | 179 | 158 | 153 | 107 | 100 | 79 | 62 | 56 | 56 | 45 | 43 | 45 | 44 | 45 | 51 | 57 | 59 | 48 | 54 | 59 | 97 | 131 | 181 |
| Protein | 55 | 65 | 68 | 67 | 68 | 54 | 60 | 56 | 64 | 62 | 67 | 58 | 59 | 60 | 61 | 60 | 67 | 66 | 71 | 69 | 65 | 67 | 69 | 72 | 69 |
| Albumin | 18 | 23 | 24 | 23 | 24 | 19 | 21 | 19 | 26 | 25 | 27 | 24 | 23 | 26 | 26 | 26 | 28 | 27 | 30 | 31 | 28 | 29 | 31 | 33 | 30 |
| Globulin | 37 | ♦42 | 44 | 44 | 44 | 35 | 39 | 37 | 38 | 37 | 40 | 34 | 36 | 34 | 35 | 34 | 39 | 39 | 41 | 38 | 37 | 38 | 38 | 39 | 39 |

- Improved ALT but reflared March 2023 when prednisone stopped
- Started on azathioprine
- Now LFTs normal

# PPIs

- Most widely used class of drugs prescribed over the long term in all of clinical medicine
- 8-10% ambulatory adults prescribed PPI in past 30 days
- In 2009, over US$13 billion spent worldwide on PPI prescriptions

- Should we be concerned about long term PPI use?

# Mechanism of action

- Inhibit H-K-ATPase in parietal cells
- Most effective when parietal cell is stimulated to secrete acid postprandially
- Amount of H-K-ATPase present in parietal cell is greatest following a prolonged fast
- PPIs should be administered before first meal of the day
- Once daily PPI dosing for 5 days inhibits maximal gastric acid output by ~66%
- Acid secretary capacity may not be restored for 24-48 hrs after discontinuing
- PPI efficacy assessed by median pH4time

# Patient WC

- 50 year old male
- Epigastric pain worse in the mornings and after meals
- Gastroscopy showed mild gastritis
- Urease test negative
- Started on omeprazole 20mg daily
- Returned after 2 weeks → no response

# Question 6- What would you do next?

- 1) Increase omeprazole to 20mg bd
- 2) Change to pantoprazole/lansoprazole
- 3) Change to famotidine
- 4) Add prokinetic

# Patient WC

- Took omeprazole 20mg bd with partial response but still ongoing symptoms
- Added in Famotidine with dramatic response
- Symptoms resolved on review 3 weeks later

# Why are some PPIs more effective in some patients than others?

- Approximately 40% of patients will not respond adequately to PPI
  - Due to Cytochrome P450 CYP2C19 genotype differences
  - Rabeprazole and esomeprazole are CYP independent
- 5% Caucasians, 12-23% Asians homozygous for CYP2C19 inactivating mutations → Delayed metabolism of PPI
- Patients homozygous for wild type gene are rapid metabolisers and have lower PPI plasma concentration
- Less likely to have successful treatment (46% success in rapid metabolisers vs 85% in normal metabolisers)

# PPIs available in NZ

Omeprazole

Pantoprazole

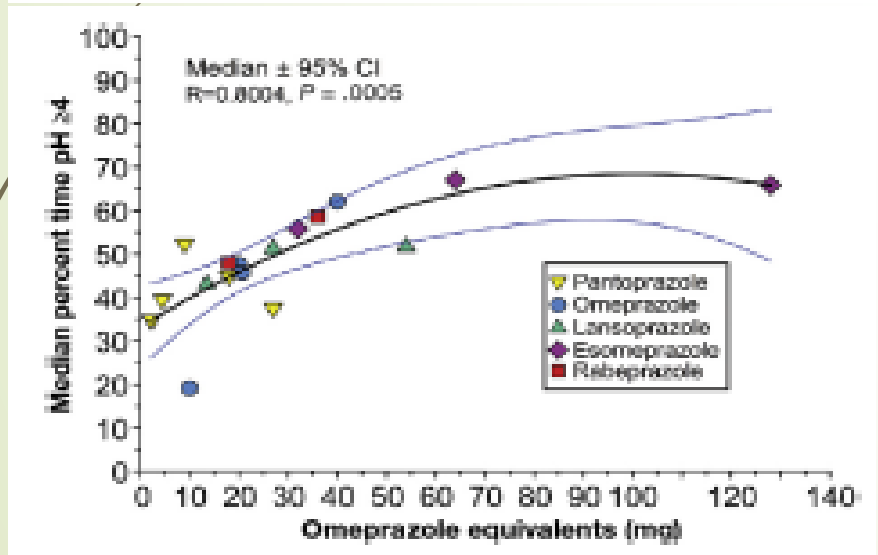
Lansoprazole

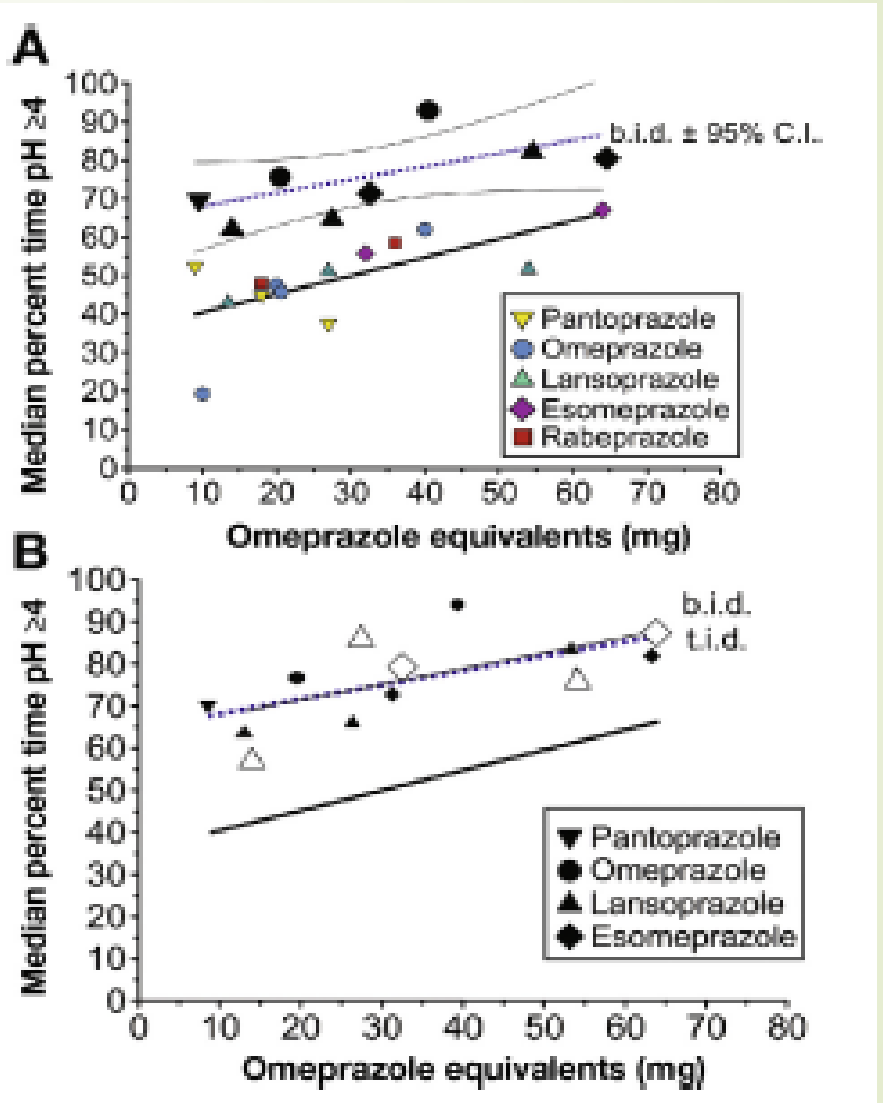
Are they equivalent?

**Table 1.** Potency of PPIs Based on OE

| Drug at lowest available dosage | OE |
|---|---|
| Pantoprazole 20 mg | 4.5 mg |
| Lansoprazole 15 mg | 13.5 mg |
| Omeprazole 20 mg | 20 mg |
| Esomeprazole 20 mg | 32 mg |
| Rabeprazole 20 mg | 36 mg |

NOTE. PPIs are listed in order of increasing potency.[17]
OE, omeprazole equivalent; PPIs, proton pump inhibitors.




Graham et al. CGH 2018

# Question 7: How do you stop acid suppression treatment?

- 1) Stop after completing course
- 2) Wean off over a few weeks
- 3) I don't tend to stop

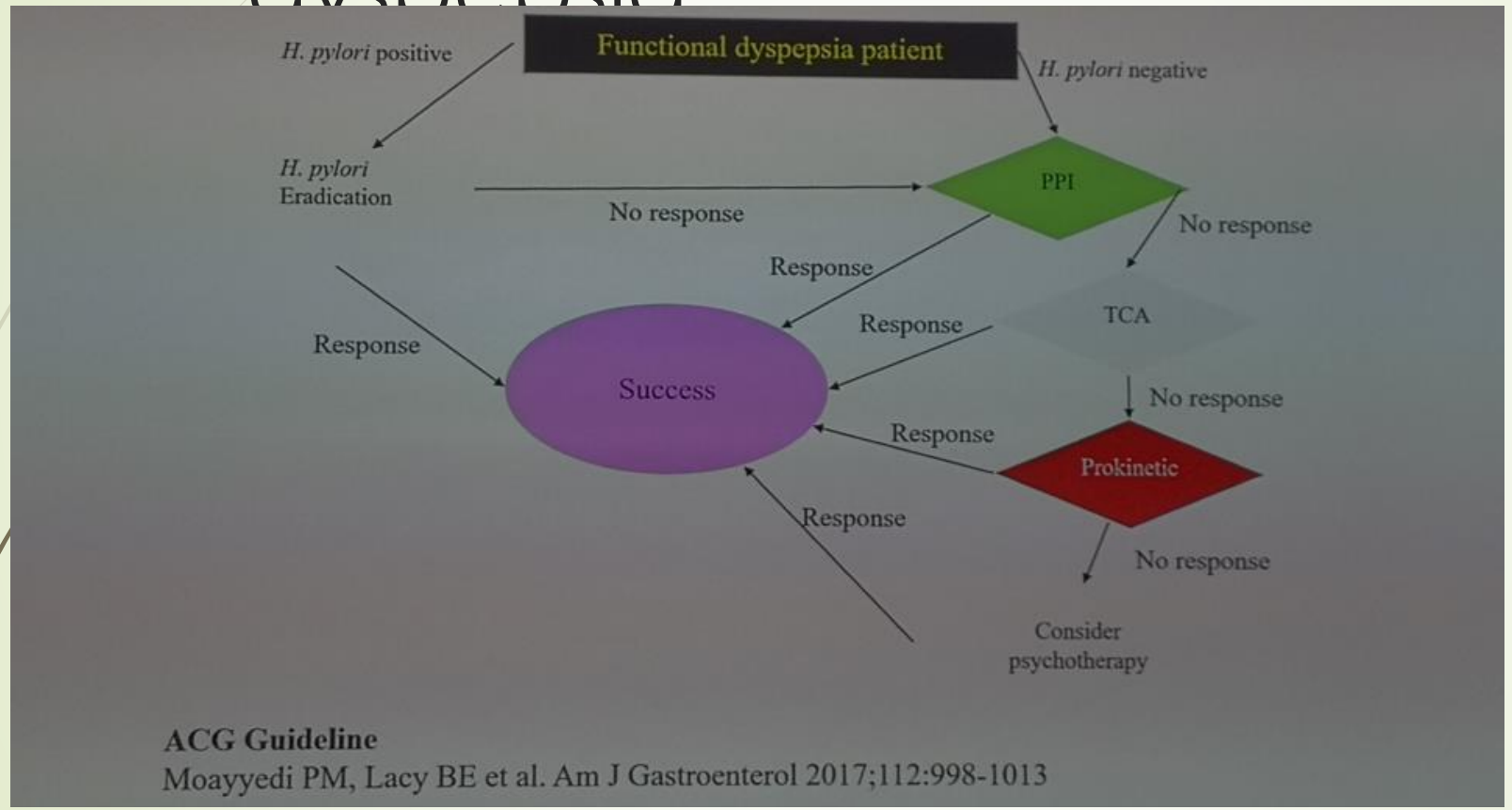
# How do I stop long term PPI therapy?

- Patients suitable to stop PPIs include functional dyspepsia, NERD (non erosive reflux disease), laryngopharyngeal reflux, no specific indication identified
- The longer a patient is on a PPI (esp if high dose), the harder it is to stop
- Suddenly stopping PPIs can result in a rebound effect- a sudden rise in gastric acid output due to hypergastrinemia
- Step down approach effective
- On-demand use can continue to be effective in many patients
- H2RAs can also be used to improve symptoms

# Question 8: What if he didn't respond to PPI/H2RA? What next?

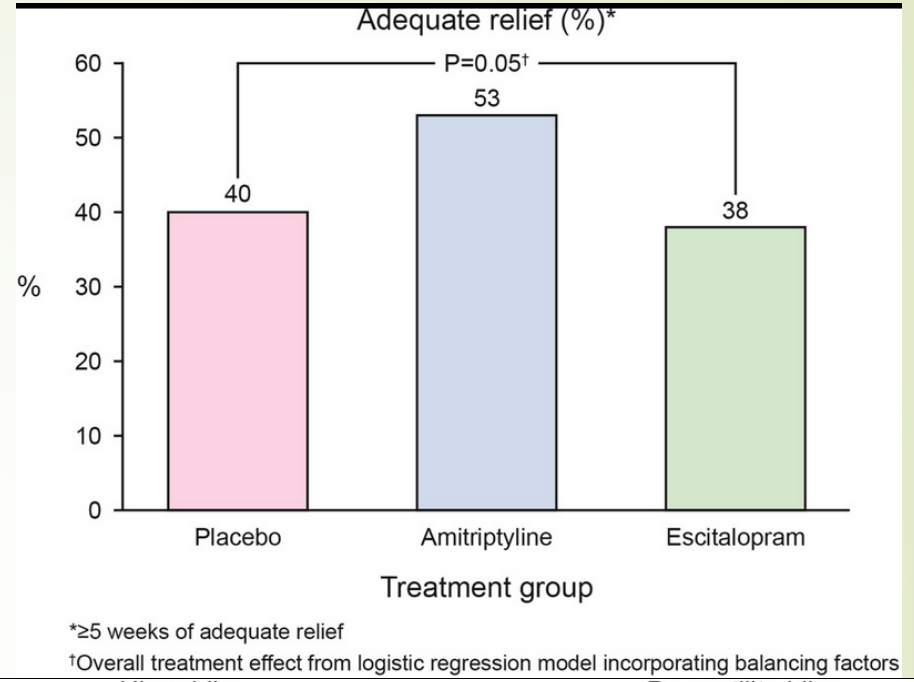
- 1) Other pain relief eg tramadol, codeine
- 2) TCA
- 3) Prokinetics
- 4) Escitalopram
- 5) Psychotherapy

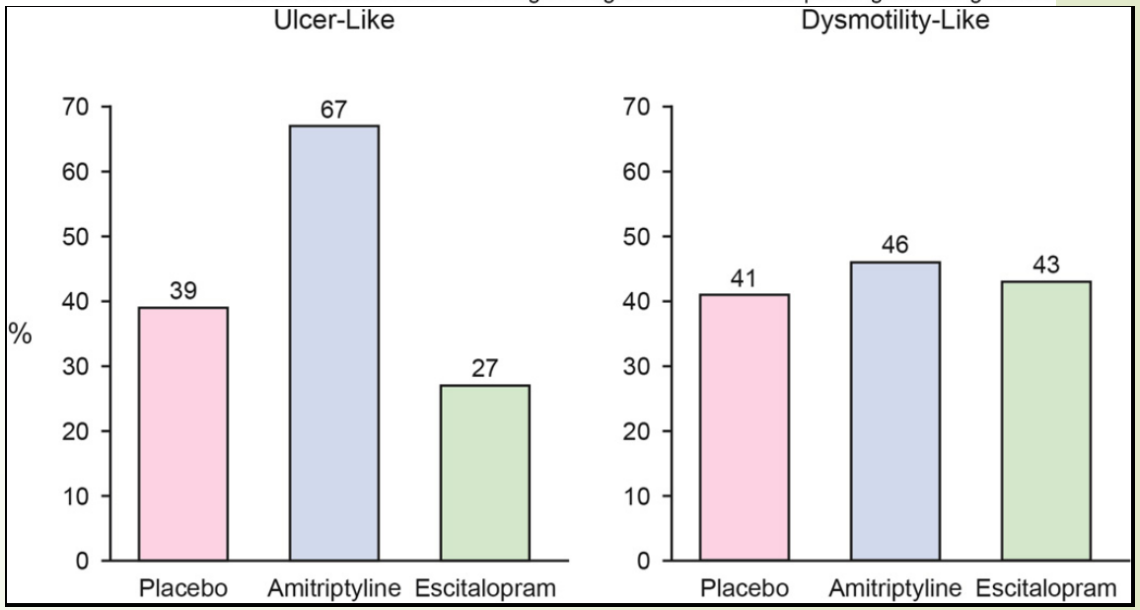
# Empirical treatment for functional dyspepsia



# Functional dyspepsia treatment trial

- Primary end point: adequate relief of overall FD symptoms
- Patients with ulcer-like FD had higher reports of adequate relief with amitryptiline
- Patients with dysmotility like FD did not respond differently




Talley et al, Gastroenterology. 2015;149(2):340-9.

# Potential adverse effects of PPI

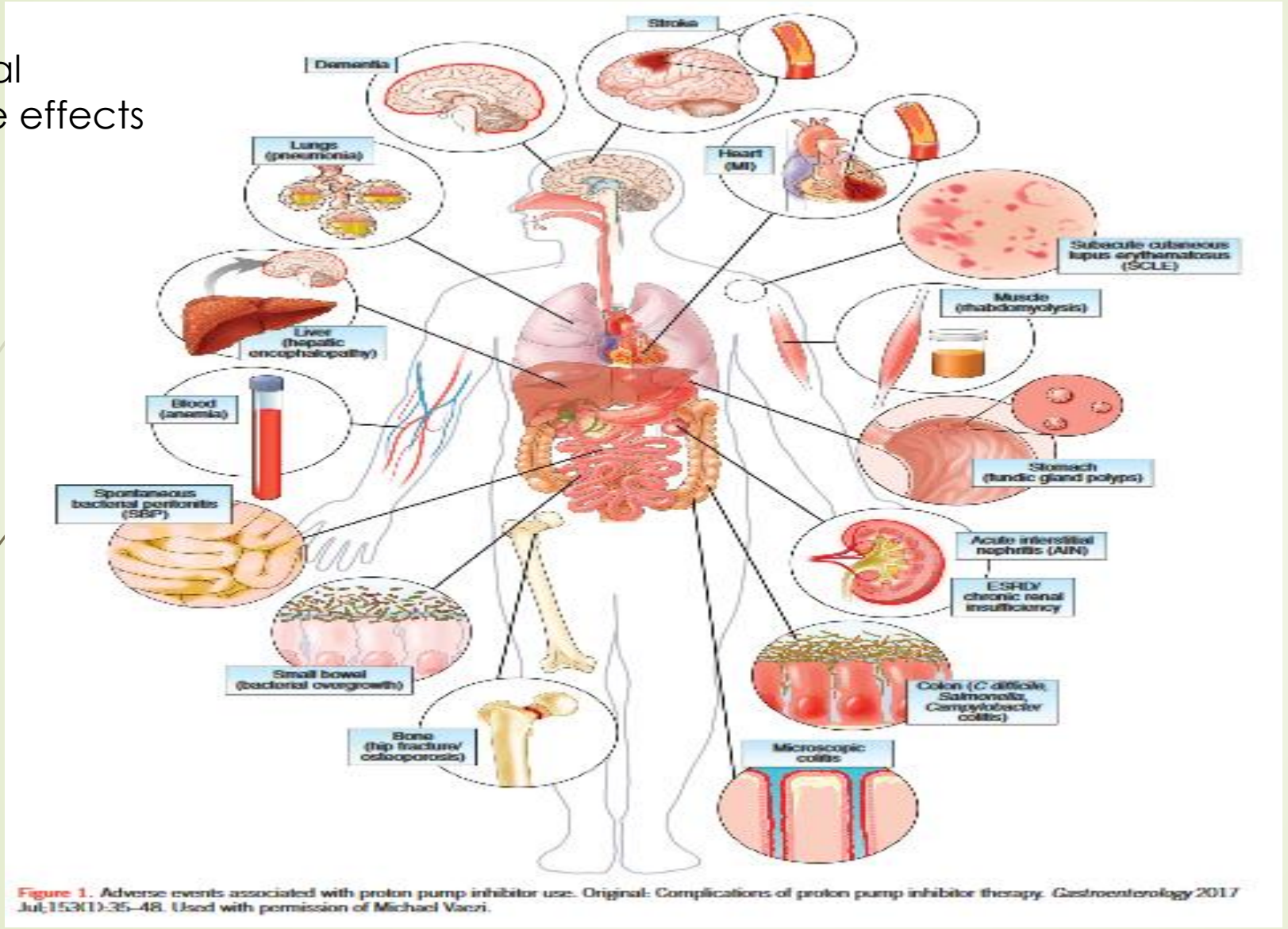



Figure 1. Adverse events associated with proton pump inhibitor use. Original: Complications of proton pump inhibitor therapy. *Gastroenterology* 2017 Jul;153(1):35–48. Used with permission of Michael Vaezi.

# Gut microbiome

- Hypochlorhydia induced by PPIs may allow survival of more organisms
- Prior studies have reported an overexpression of oral bacteria in the faeces of individuals taking PPI
- Increased ratio of Firmicutes to Bacteroidetes at the phyllum level

# Infection

- Reduction of acidity may allow survival of microbes especially acid sensitive organisms eg vibrio cholera, salmonella, Campylobacter
- PPIs may also increase intestinal permeability
- Numerous observational studies report an association between PPIs and C.diff infection but a causal link has not been demonstrated

# Chronic Kidney Disease

- Weak data
- Recent meta-analysis shows weak association, could not exclude residual confounding
- When compared to non PPI users, pooled risk of CKD with PPI= 1.2
- When compared to H2RAs, pooled risk = 1.29

Wijarnpreecha Dig Dis Sci 2017;62:2821-7

# Cognitive decline

- Concern regarding increased risk of cognitive decline came from a German cohort study on health in aging, which assessed nursing home patients
- Four recent studies showed no risk of cognitive decline [1,2,3,4]
- Large cohort studies including Nurses Health study, US population based cohort study[5] and a very large Finnish nested case-control study[6] have shown **no significant association** between PPI use and Alzheimer's disease


1 Lochhead Gastroenterology 2017
2 Wod Clin Gastroenterology and Hepatol 2018
3 Gray J Ann Ger Society 2017
4 Taipale Am J Gastroenterol 2017
5 Gray SL J Am Geriatri Soc 2017
6 TaipalAm J Gastroenterol 2017

# Myocardial infarction and stroke

- Meta-analysis of 17 GORD studies showed HR of 1.7 for patients taking PPIs, but quality of evidence was low
- Danish cohort study of 214998 patients reported minimal increased incidence in ischemic stroke and MI in PPI users (HR 1.13 and 1.31) but confounded by variables (smoking, obesity, exercise status)
- Two large American cohort studies have shown no increased risk of MI in PPI users

# Osteoporosis/Fractures

- Retrospective cohort study showed association of PPI use with osteoporosis and hip fracture in stroke patients[1]
- Systematic review reported significant association between PPI use and hip fracture (PPI users have 26% higher risk of hip fracture than non-PPI users)[2]

1 Lin SM Osteoporosis Int 2018
2 Hussain, Rheumatol Intl 2018

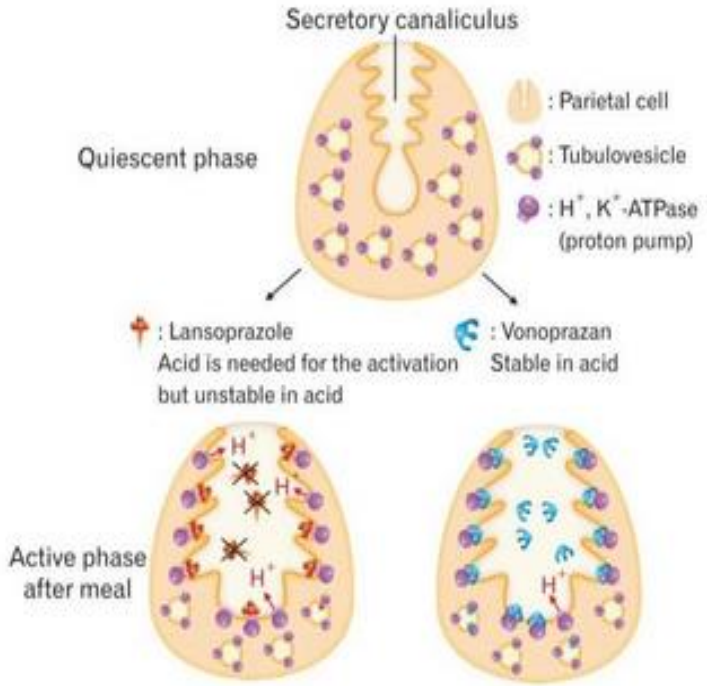
# Who should remain on long term therapy?

- Treatment of erosive esophagitis (LA Grade C and D). Risk of relapse is 72% if PPI is stopped
- PPI responsive esophageal eosinophilia
- High risk, long term NSAID users
- Barretts esophagus
- Zollinger-Ellison syndrome

# P-CABs Mechanism of Action

## P-CABs

- Rapidly and reversibly inhibit the proton pump preventing acid production
- Able to elevate gastric pH to a higher level than PPIs
- Have dose-dependent effects on acid production
- Exert full effect from the first dose




Vonoprazan stably accumulates in the acidic secretory canaliculus and noncovalently binds to proton pump with a very slow dissociation rate and can inhibit newly exposed proton pump for a long time.

Oshima T, et al. J Neurogastroenterol Motil. 2018;24:334-344; Scarpignato C, et al. Aliment Pharmacol Ther. 2015;42:1027-1029.

# Potassium-competitive acid blockers

- Licensed in some Asian and South American countries
- Faster onset of action and more profound control of acid secretion than PPIs
- Examples = revaprazan, vonoprazan, tegoprazan and fexuprazan



New Zealand Government

# UPDATE ON POLYP SURVEILLANCE GUIDELINES

2024

# POLYP RISK CATEGORIES

**Table 2: Polyp risk categories**

| | Conventional adenomas | Serrated polyps |
|---|---|---|
| Average-risk polyps | Tubular adenomas < 10 mm | Sessile serrated lesion (SSA/P) < 10 mm<br>Hyperplastic polyp ≥ 10 mm* |
| High-risk polyps | Adenoma ≥ 10 mm**<br>Adenoma with tubulovillous or villous histology**<br>Adenoma with high-grade dysplasia** | Sessile serrated lesion (SSA/P) ≥ 10 mm<br>Sessile serrated lesion (SSA/P) with dysplasia<br>Traditional serrated adenoma<br>Serrated adenoma, unclassified (unclassified serrated polyp with dysplasia) |

* Follow up as a high-risk polyp if concern exists about consistency in distinction between sessile serrated lesion and hyperplastic polyp locally.

** Advanced adenoma defined as: 10 mm or larger in size or 25% or greater villous histology (that is, tubulovillous or villous adenoma) or high-grade dysplasia.

After piecemeal resection of polyps **equal to or greater than 20 mm** in size, the site should be checked within two to six months and then a further full colonoscopy performed after an additional 12 months. Once no recurrence is confirmed patients should undergo post polypectomy surveillance after a further interval of three years. The need for further surveillance should then be determined in accordance with this update and individual risk factors.

No surveillance is required for anyone who has only hyperplastic polyps smaller than 10 mm, unless the person meets the criteria for Serrated Polyposis Syndrome.

**Figure 1: Surveillance intervals based on findings at high-quality colonoscopy**

| 1 year | 3 years | 5 years | 10 years or NBSP (Whichever comes first) |
|---|---|---|---|
| **Adenomas*** | **Adenomas*** | **Adenomas*** | **Adenomas*** |
| ≥10 adenomas*** | 5–9 adenomas <10 mm<br>Adenoma ≥10 mm<br>Tubulovillous adenoma or Villous adenoma<br>Adenoma with HGD | 3–4 adenomas <10 mm | 1–2 adenomas <10 mm |
| **Serrated polyps*** | **Serrated polyps*** | **Serrated polyps*** | |
| Serrated polyposis syndrome – initial interval after polyp clearance*** | ≥5 SSL <10 mm<br>SSL ≥10 mm<br>SSL with dysplasia<br>Traditional serrated adenoma | 1–4 SSL <10 mm<br>HP ≥10 mm** | |

* If there are both adenoma <10 mm and SSL <10 mm, the numbers should be summed up and follow-up interval for SSL should be applied.

** A 3-year follow-up interval is favoured if concern about consistency in distinction between sessile serrated lesion and hyperplastic polyp locally.

*** Consider referral to NZ Familial Gastrointestinal Cancer Service (NZFGCS), see referral criteria below .

**NBSP**: National Bowel Screening programme

**SSL**: Sessile Serrated Lesion (= Sessile Serrated Adenoma/ Polyp)

**HGD**: High Grade Dysplasia

**HP**: Hyperplastic Polyp

* If there are both adenoma <10mm and SSL <10 mm, sum up the numbers and apply follow-up interval for SSL.

** A three-year follow-up interval is favoured if concern about consistency in distinction between sessile serrated lesion and hyperplastic polyp locally.