MedNut Mail

The How, When, Where, Which and Why of pharmacotnutrition

Calcium and proton pump inhibitors

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Commentary

Calcium carbonate is a commonly prescribed therapeutic intervention and is listed on the Pharmaceutical Benefits Scheme (PBS). However, there is increasing concern regarding the therapeutic effectiveness of calcium carbonate when there is concurrent administration of a proton pump inhibitor (PPI).

Calcium is primarily absorbed throughout the intestines and predominantly from the duodenum and jejunum.

Calcium carbonate.

Calcium carbonate is an insoluble salt which means it requires a lower pH (closer to 1) for calcium to be split from the carbonate. There is reduced availability of calcium from calcium carbonate in the presence of PPIs due to the change in pH ie PPIs raise the pH closer to 7 and consequently smaller quantities of absorbable calcium are released from the carbonate.

Calcium carbonate may be administered to those prescribed PPIs longterm, however the calcium carbonate must be taken either with meals or immediately after meals, to take advantage of the meal-induced acid production, however, the amount of calcium absorbed may still not be adequate for body requirements. If administered whilst fasting ie on an empty stomach, calcium absorption from calcium carbonate is minimal.

Calcium citrate.

Calcium citrate is a soluble salt form which means it does not require gastric acidity to enhance or support calcium absorption.

Calcium citrate absorption is also not impacted by presence or absence of food.

Because calcium citrate reduces parathyroid hormone (PTH) activity there is reduced bone resorption.

As calcium citrate is not dependent on acid or low pH (closer to 1) for absorption, it may be the preferred calcium supplement for PPI users.

Calcium in natural products such as cheese and milk, will provide greater calcium availability regardless of pH. Further, evidence indicates PPIs do not reduce the absorption of calcium contained in milk and cheese, presumed as a consequence of the “meal effect”.

Ultimately which calcium intervention is recommended seems to be a cost issue – calcium carbonate is registered on the PBS and therefore the cost is minimal, whilst calcium citrate is not registered on the PBS and so the consumer pays the full price. The

trade-off seems to be cost versus benefit ie if PPI consumers prescribed calcium carbonate do not understand the importance of taking their calcium carbonate with, or immediately after, meals then there is minimal cost and minimal benefit.

Given the rapidly increasing numbers of prescriptions for PPIs the question needs to be asked *why isn't calcium citrate also registered for the PBS?* The prescription of calcium carbonate without adequate emphasis on timing of consumption means it is an ineffective intervention ie looks as though something is being done without actually conferring any benefit or phrased more bluntly – a waste of time, effort and money.

What actions will you initiate when you see someone prescribed both calcium carbonate and a PPI – will you -

- check that the calcium carbonate supplement is being consumed during or immediately after meals?
- recommend a change to calcium citrate to ensure adequate calcium absorption?
- make recommendations to increase consumption of more high calcium food sources in order to maximise calcium absorption?

Conclusions

PPIs increase gastric pH closer to neutral ie less acidic, and most of the evidence seems to confirm they may negatively affect the calcium absorption from calcium carbonate. There are some effective strategies to ensure an adequate intake of calcium in the presence of a PPI that include an alternate calcium salt, timing of calcium intake, and increasing intake of high-calcium foods.

Case study

Medical History with Nutritional Aspect

Amputation	☐	Constipation	☑	Dysphagia	☐	MND	☐
Anaemia	☐	CVA	☐	Enteral Feed	☐	MS	☐
Arthritis	☑	CVD	☐	Falls	☐	Osteoporosis	☐
Cancer	☐	Dementia	☑	Fracture	☐	PD	☐
CCF	☐	Dentures	☐	Frailty	☐	Pressure Area	☐
Chest Infection	☐	Depression	☑	Gout	☐	Renal	☐
COAD	☐	DM Type 1	☐	Hypertension	☑	Ulcer	☐
Confusion	☐	DM Type 2	☐	Incontinent	☐	UTI	☐

Food Allergies: reduced sense of smell, oedema

Other: chronic pain, anger and irritiability, AF, DVT

Biochemistry with Pharmaconutritional Consequences

Na:	141	mmol/l	Hb:	112	g/L	Albumin:	29	g/L	BSL:		mmol/l
K:	4.5	mmol/l	Lymph:	1.0		Total Protein:	61	g/L	HbA1C:		
Urea:	4.7	mmol/l	MCV:	95	mmol/l	B12:		pmol/L	INR:		
Creatinine:	0.079	mmol/l	Zn:		umol/l	Folate:		nmol/L	TSH:		mIU/L

Other: eGFR 59, CRP 45

Medications That May Adversely Affect Nutritional Status

Drug	Vits + Mins	bpp	>90%	N/V	C/D	Wt	App	Tst	Thir	Sal	Drlg	d m	Dys	BSL
COLOXYL WITH S			☐		D			☐				☐	☐	☐
COVERSYL			☐	NV	D			☑				☑	☐	☐
Digoxin	Mg		☐	NV	D		↓	☐				☐	☐	☐
PANADOL			☐	NV	CD			☐				☐	☐	☐
XARELTO			☑	NV	CD			☐				☑	☐	☐
			▩					▩				▩	▩	▩

Extra drug:

Comments – medication and nutrition impacts (direct and indirect) only

Relatively recent available relevant biochemistry indicates

- low albumin and marginal total proteins – may alter Xarelto effect due to it's > 90% binding to plasma proteins.

Chronic use of coloxyl + senna may promote excessive loss of water and electrolytes, especially potassium, and their regular monitoring recommended.

Coversyl impairs zinc status.

Digoxin increases urinary excretion of magnesium – advisable to check status.

Dietary levels of caffeine intake in conjunction with paracetamol inhibit antinocieception.

Concurrent ingestion of panadol and iron resulted increased rate of iron absorption and decreased extent of drug absorption therefore advisable for drug and iron to be administered at different times from each other to reduce risk of drug-nutrient interaction.

Staff advise Mrs AAX was a good eater and now eats minimally, drinks even less, and often clamps her mouth shut; she is fully assisted with her meals.

Mrs AAX is a small, pale, frail lady with a lovely smile and who was drinking a glass of water when I went to see her - she responded to my presence.

Pharmaconutrition factors that may be impacting on food intake include

- sub-therapeutic digoxin levels - they feel unwell and so don't eat. There is disagreement between pathology labs with regard to acceptable lower digoxin level with some labs stipulating 0.8 units and others stipulating 0.6 units; personal experience indicates 0.8 units is the best level to be guided by ie if digoxin levels below 0.8 units then review of intervention recommended.

Mrs AAX's diagnoses include chronic pain. Pharmaconutrition factors that may be useful to consider in pain management include -

- magnesium – proposed mechanism magnesium blocks the NMDA receptor channels in the spinal cord and thus limits the influx of calcium ie reduces the risk of excitotoxicity and consequent exacerbation of pain. Currently prescribed digoxin which increases magnesium excretion.

What else would you include?

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