MedNut Mail

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

Costs of inaction

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19th July 2022

https://medicationsandnutrition.online

Commentary

Costs of inaction is a term typically associated with business decisions – what are the costs if you choose not to purchase this product or that service - would there be loss of revenue, loss of opportunity, loss of expansion, increased risk of harm in some form.

If we apply *costs of inaction* to the health sector then the costs are different and usually more profoundly detrimental as they impact care recipients, their families and consequently society. For example, in the late 1800s the "cornbelt" area in America was known for the 4 x Ds ie dermatitis, diarrhoea, dementia and death. In the late nineteen teens, a researcher discovered niacin (vitamin B3) and proved niacin deficiency was the cause of the 4 x Ds, however nothing was done until America decided to send troops to Europe in the 1939-1945 war.

Imagine the cost of that inaction especially during the 20-year period between discovery and intervention – the loss of life in the millions, the negative impacts on families due to the loss of one or more family members (emotional, financial, etc), the loss to society in productivity and contribution – these *costs of inaction* would have been immense.

Are we already ascribing many symptoms to disease progression that may not be related to the actual disease but rather are treatment-induced? Similar to the niacin example, is that because what we see we consider to be "normal" and therefore not treatable?

That the negative consequences of pharmaconutrition are still not integral components of nutrition research, nor included in nutrition journals and conferences is scary … and the oversight further enhances the *costs of inaction*.

If you are reading this then it is likely you are an early adopter – a person who is more likely to try new ways of doing things to improve the outcomes for those in your care –

- do you regularly review your clinical practice, and especially the pharmaconutrition aspect of your practice, in order to minimize your *costs of inaction* to those in your care?
- have you integrated pharmaconutrition into all aspects of your daily clinical practice such as assessment forms, clinical reports, entries in the Notes?

Conclusions

Increasingly the research evidence of xenobiotic (pharmaceutical, toxic metal, other) inhibition of physiological transporters is inferring a likely pharmaconutrition contribution to nutritional harm. The *costs of inaction* in disregarding the evidence or delaying initiation of action are/will result in sustained nutritional harm to millions of people globally.

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: schizophrenia

Other: blindness, STML, hypokalaemia, AF, GORD

Biochemistry with Pharmaconutritional Consequences

No recent relevant biochemistry available.

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
Aspirin	C, Fe	☑	NV				☐				☐	☐	☑
DURO-K		☐	NV	D			☐				☐	☐	☐
Frusemide	(80 mg/day) Ca, Cl, K, Mg, Na,	☑	NV	CD		↓	☐				☑	☐	☑
Metoprolol		☐	NV	CD	↑		☑				☑	☐	☑
MOVICOL		☐	N	D			☐				☐	☐	☐
Olanzapine		☑		C	↑	↑	☐				☑	☐	☐
Pantoprazole	(40 mg/day) B1, B12, Ca, Fe,	☑	NV	CD		↓	☑				☑	☐	☐
SIGMAXIN	Mg	☐	NV	D		↓	☐				☐	☐	☐
Venlafaxine		☐	NV	CD	↕	↓	☑				☑	☐	☐
		▩					▩				▩	▩	▩

Extra drug:

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

No recent relevant biochemistry available. Advisable to check plasma proteins (albumin, total proteins) as they are the primary transporters for five of the prescribed drugs and hypoproteinaemia may alter their effects and side effects.

Since Mr ABU's diagnoses includes hypokalaemia, and currently prescribed Duro-K, digoxin and frusemide, advisable to check potassium levels and clarify status.

Six prescribed medications include nausea and vomiting as side effects.

Currently prescribed four medications that may alter glycaemia, being aspirin, frusemide, metoprolol and olanzapine.

Vitamin C (960 mg/day) attenuates aspirin-induced gastric injury.

Frusemide increases urinary excretion of calcium, magnesium, potassium, sodium and thiamine.

Pantoprazole decreases B12, vitamin C, magnesium, zinc and iron absorption, may decrease calcium absorption, and decreases thiamine availability.

Digoxin increases urinary excretion of magnesium.

Three drugs alter magnesium availability - one decreases absorption and two increase urinary excretion - magnesium is important in muscle function, especially cardiac muscle amongst other functions. Magnesium deficiency manifests as confusion, disorientation, personality changes, loss of appetite, depression, muscle cramps, tingling, numbness, hypertension, cardiac dysrhythmia, seizures. Magnesium is an intracellular ion therefore serum levels are unlikely to detect early depletion of status - advisable to check magnesium levels and if low then intervention recommended. Men require 420 mg magnesium per day; the Upper Limit for magnesium from non-food sources is 350 mg elemental magnesium per day. If an intervention is commenced then advisable to consider one that provides about 300 mg elemental magnesium per day, however oral magnesium interventions are unlikely to be effective whilst pantoprazole prescribed.

Bowels –

- regular aperient prescribed,
- no PRN interventions prescribed,
- Nurse Initiated oral interventions administered 2 x Oct, 2 x Sep, 1 x Aug.

Staff advise Mr ABU eats very well.

Mr ABU is a tall, slender, charming man who told me he eats well and has no complaints about the food.

Mr ABU is both a coffee drinker and prescribed aspirin – it is likely his coffee intake is quite regular in timing and quantity and therefore having a similar day-to-day impact on aspirin effect.

Inadequate B12 status is associated with both blindness and incontinence therefore advisable to check B12 levels.

Commencement of pantoprazole indicates prudent clinical practice for B12 management as outlined:-

- establish B12 status at commencement of drug treatment, and monitor on a regular basis, or
- commence a prophylactic B12 intervention with oral supplements as they are not protein-bound and therefore do not require gastric acidity for absorption.

Given neither of these prudent clinical practices have been initiated advisable to clarify B12 status and if low then intervention recommended.

Mr ABU has a non-healing toe wound that he picks at - nutritional interventions that support to wound healing include -

- adequate status of B12, magnesium, zinc and iron - pantoprazole is prescribed which compromises their status;
- adequate vitamin C status - important in collagen formation and the strength of the collagen; both topical application and increased oral intake confer benefit. Currently prescribed pantoprazole which reduces availability of active vitamin C. It is also likely vitamin C interventions are unlikely to be effective whilst a proton pump inhibitor is prescribed.

Olanzapine, metoprolol and pantoprazole all inhibit thiamine uptake which means limited organ access and possibly elevated blood levels.

What else would you include?

Medications have profoundly and positively changed health outcomes however they do generally come with some nutritional harms. By identifying and addressing the nutritional harms, optimal health outcomes are closer to being achieved.

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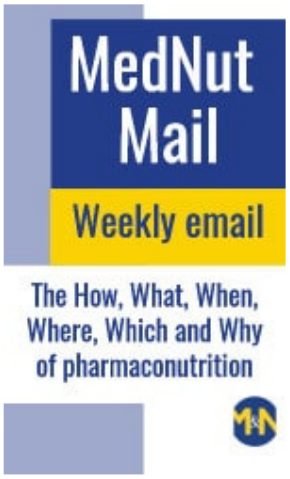

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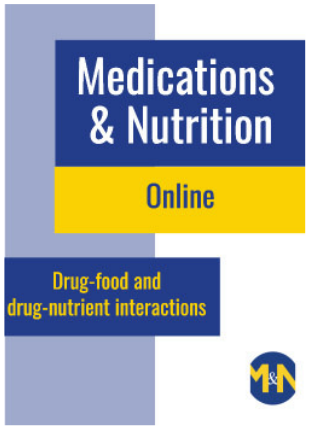

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