

MedNut Mail

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

Iron supplements – the bad and the ugly

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<https://medicationsandnutrition.online>

Commentary

Iron supplements are generally prescribed if serum iron levels are low and there is rarely any further intervention if iron status does not improve. Apart from possible “bleeds” we do not typically consider other causes of low iron status such as:

- **lack of nutrients necessary for haemoglobin formation.**

Examples include

- **Biotin** - anti-epilepsy medications such as carbamazepine and sodium valproate decrease biotin absorption – biotin is important in 5 stages of haemoglobin formation,
- **Riboflavin** – some antihypertensives such as amlodipine, atenolol, metoprolol, perindopril and antipsychotics such as prochlorperazine decrease riboflavin absorption – riboflavin is important in haemoglobin formation.

- **acid-inhibiting medications decreasing iron absorption.**

Esomeprazole increases gastric acidity pH to > 4, pantoprazole possibly even higher, lansoprazole > 3.

Combination Ferrous and Vitamin C

Iron is available in two states –

- **ferrous** - primarily available in animal foods, and
- **ferric** - primarily available in plant foods.

Administration of vitamin C with plant foods increases the availability of iron by converting ferric to ferrous ie the absorbable form of iron.

Combination ferrous + vitamin C is a matrix product and consequently the duration to complete dissolution is more than 4 hours at a pH of 1.2; administration of each nutrient separately reduces duration of complete dissolution to less than one hour at a pH of 1.2.

The iron in iron + vitamin C combination tabs is in the ferrous state and therefore administration with vitamin C does not confer physiological advantage – perhaps the primary benefit is a financial one for the manufacturers.

Impact on gut microbiota

Poorly absorbed iron, typically from sources such as iron supplements, results in increased iron availability to the gut microbiota with consequent:

- increase in the virulence of faecal entero-pathogens,
- increase in the ratio of faecal entero-pathogens to protective microbiota such as bifidobacteria and lactobacillus,
- preferential colonisation of the gut microenvironment by potentially pathogenic strains, and
- increased gut inflammation.

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The growth and colonisation of Lactobacillus and other similar (beneficial) bacteria is not dependent upon the availability of iron. Lactobacillus and other similar bacteria provide an important barrier effect against colonisation and invasion by pathogens – iron fortification may result in increased pathogen representation and consequently dilute the number of beneficial bacteria and thus weaken the protective effect they confer.

Evidence indicates beneficial gut microbiota have low iron requirements and that entero-pathogens thrive once iron is available. Non-haem iron, such as iron from supplements, is poorly absorbed therefore there is an increased availability of iron for the entero-pathogens which increases the risk of gastro-intestinal infections. If iron is administered attached to a protein such as in animal foods, then

iron absorption is improved x 10 and, potential increase in pathogen numbers and consequent harm is reduced.

A strategy to minimise potential harm caused by administration of iron supplements is to administer them concurrently with a probiotic to support the beneficial bacteria ratio.

Iron supplements can cause harm by altering gut microbiota in favour of pathogens, absorption can be modified by gastric acidity pH, and administration with vitamin C is monetising ignorance. Consequently administration of iron interventions requires consideration for both type of intervention and its duration in order to optimise their effectiveness.

Do you review duration and effectiveness of iron supplements in your clinical practice?

Case study

Medical History with Nutritional Aspect

Amputation	<input type="checkbox"/>	Constipation	<input type="checkbox"/>	Dysphagia	<input type="checkbox"/>	MND	<input type="checkbox"/>
Anaemia	<input checked="" type="checkbox"/>	CVA	<input type="checkbox"/>	Enteral Feed	<input type="checkbox"/>	MS	<input type="checkbox"/>
Arthritis	<input type="checkbox"/>	CVD	<input checked="" type="checkbox"/>	Falls	<input type="checkbox"/>	Osteoporosis	<input type="checkbox"/>
Cancer	<input type="checkbox"/>	Dementia	<input type="checkbox"/>	Fracture	<input type="checkbox"/>	PD	<input type="checkbox"/>
CCF	<input checked="" type="checkbox"/>	Dentures	<input type="checkbox"/>	Frailty	<input type="checkbox"/>	Pressure Area	<input type="checkbox"/>
Chest Infection	<input type="checkbox"/>	Depression	<input type="checkbox"/>	Gout	<input checked="" type="checkbox"/>	Renal	<input checked="" type="checkbox"/>
COAD	<input checked="" type="checkbox"/>	DM Type 1	<input checked="" type="checkbox"/>	Hypertension	<input checked="" type="checkbox"/>	Ulcer	<input type="checkbox"/>
Confusion	<input type="checkbox"/>	DM Type 2	<input type="checkbox"/>	Incontinent	<input type="checkbox"/>	UTI	<input type="checkbox"/>
Food Allergies:	<input type="text" value="hemicolectomy, intermittent chronic diarrhoea"/>						
Other:	<input type="text" value="chronic back pain, SOB, vit D def, B12 def, GORD"/>						

Biochemistry with Pharmaconutritional Consequences

No recent relevant data available

Medications That May Adversely Affect Nutritional Status

Drug	Vits + Mins	bpp >90%	N/V	C/D	Wt	App	Tst	Thir	Sal	Drig	d m	Dys	BSL
Allopurinol		<input type="checkbox"/>	NV	D			<input checked="" type="checkbox"/>				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Furosemide	(120 mg/day) Ca, Cl, K, Mg, Ni	<input checked="" type="checkbox"/>	NV	CD		↓	<input type="checkbox"/>				<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input checked="" type="checkbox"/>
HYDREA		<input type="checkbox"/>	NV	CD	↕	↓	<input type="checkbox"/>				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Nebivolol		<input checked="" type="checkbox"/>	NV	CD			<input type="checkbox"/>				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Paracetamol		<input type="checkbox"/>	NV	CD			<input type="checkbox"/>				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Perindopril		<input type="checkbox"/>	NV	D			<input checked="" type="checkbox"/>				<input checked="" type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
SPREN	C, Fe	<input checked="" type="checkbox"/>	NV				<input type="checkbox"/>				<input type="checkbox"/>	<input type="checkbox"/>	<input checked="" type="checkbox"/>
		<input checked="" type="checkbox"/>					<input checked="" type="checkbox"/>				<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>	<input checked="" type="checkbox"/>
Extra drug:	<input type="text" value="lantus 40U bd, novorapid 30U/18U/ 8U"/>												

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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 3 of the prescribed drugs and hypoproteinaemia may alter their effects including expression of their side effects.

Insulin regimen

- Breakfast - novorapid 30U, lantus 40U
- Midday - novorapid 18U
- Evening - novorapid 8, lantus 40U

Diabetes drugs

- lantus has a time to onset of 1hour, minimal peak, and duration of 20-26 hours,
- novorapid has a time to onset of 15 minutes, 30-90 minutes time to peak, and duration of 3-5 hours.

Diabetes drugs coverage

- **before breakfast BSLs** - minimal if any coverage from previous morning's lantus; covered by previous evening's lantus;
- **before midday BSLs** - covered by previous evening's lantus, some coverage from current morning's novorapid; covered by current morning's lantus;
- **before evening meal BSLs** - minimal coverage from previous evening's lantus and current midday's novorapid; covered by current morning's lantus;

- **before supper BSLs** - minimal coverage from previous evening's lantus; covered by current morning's lantus and current evening's lantus and novorapid.

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

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

Perindopril impairs zinc status - zinc is important in immune function, glycaemic control, and sense of taste amongst other functions.

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

At increased risk of thiamine and choline deficiencies as nebivolol inhibits their physiological transport therefore interventions recommended, and advisable to administer either one hour before or 2 hours after drug administration.

If a thiamine intervention is initiated then advisable to consider a magnesium intervention (to provide ~ 300 mg elemental magnesium/day) as adequate magnesium status is essential for activation of thiamine, vitamin D and iodine.

Mr AAD commented the food has no taste; he is currently prescribed frusemide. Drugs such as frusemide significantly increase zinc excretion - zinc is important in sense of taste, release of the hunger hormone

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Neuropeptide Y, and insulin production amongst other functions. Advisable to check zinc levels and if low then consider a short term (90-120 days) intervention.

Many of Mr AAD's diagnoses fall under the dysfunctional mitochondria umbrella - diabetes, gout,

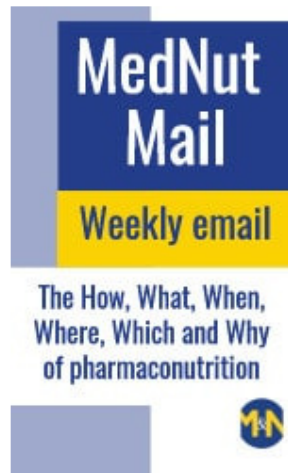
hypertension, CVD, etc which means there is a likely increased demand on nutrient availability - thiamine is a particular nutrient of concern because it is at increased risk of inadequacy due to the direct and indirect impacts of nebivolol and frusemide.

What else would you include?

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