

MedNut Mail

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

Deafness and pharmacotnutrition

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1st February 2022

<https://medicationsandnutrition.online>

Commentary

Deafness is a multifactorial, global health issue that is commonly inadequately managed in healthcare services; for example a person who responds inappropriately to a question is automatically labelled “? dementia” and no-one even considers the alternative ie “? deafness”.

When a deaf person does not respond appropriately, even when spoken to with a raised voice, then the clinician typically gives up trying to communicate which leaves the deaf person feeling diminished and not worth any effort ie a third-class citizen. Sadly, this is a very common scenario. Tip – carry a small portable whiteboard and pen – you write and they can reply verbally – useful strategy for minimizing communication confusion, and doesn't cost much extra time.

Is there evidence of an association between deafness and nutrition factors? Surprisingly, given the extent of deafness globally, this area has attracted very little attention. Outlined are a number of key nutrition factors for which there is evidence -

B12 and folate

Low B12 and/or folate have been found to be associated with deafness, and in fact some researchers recommended vitamin B12 and folate

testing be routinely included when evaluating symptomatic hearing loss.

There is some evidence that B12 interventions are therapeutic in tinnitus management.

Vitamin C

Hearing impairment is being related to inadequate dietary intake especially in the elderly, particularly with findings that an adequate dietary intake of vitamin C is associated with better hearing in the older population.

Vitamin D

Low vitamin D status was associated with low-frequency and speech-frequency hearing loss in the elderly which indicates low vitamin D status may be a potential risk factor for age-related hearing impairment.

Early evidence suggests early correction of vitamin D deficiency in newly-diagnosed Meniere's disease reduces the necessity for more extreme interventions.

Iron Deficiency Anaemia (IDA)

Evidence indicates mild maternal IDA during pregnancy and lactation may inhibit hair cell development in the infant.

Further, latent iron deficiency is associated with abnormal auditory neural maturation in infants at 34 weeks gestational age.

Zinc

Inadequate zinc status has been associated with impaired hearing.

Coffee

Evidence indicates coffee protects against hearing loss and tinnitus.

Polyunsaturated Fatty Acids (PUFAs)

Evidence indicates an inverse association between long-chain n-3 PUFAs and hearing impairment.

Thiamine

There is some recent evidence that thiamine deficiency can present as bilateral hearing loss.

Thiamine transporter OCT2 is expressed in the hair cells of the cochlea therefore interruptions to thiamine accessibility are likely to impact hair cell function.

Dysfunctional mitochondria

The evidence is increasing that diagnoses that include dementia, diabetes, overweight and/or obesity ie all diagnoses associated with dysfunctional mitochondria, are associated with increased risk of hearing impairment.

Thyroid function

A fundamental function of T3 is to stimulate sensory development – an essential means for acquiring information from our environment.

Frailty

There is some evidence of an inverse correlation between hearing impairment and frailty. Further, moderate or greater hearing impairment in older adults is associated with decreased levels of physical activity.

There is some interesting evidence that fenofibrate protects against cisplatin-induced ototoxicity by maintaining peroxisome and mitochondria number and function, reducing inflammation, and decreasing Reactive Oxygen Species levels.

Apoptosis (programmed cell death) is a calcium-dependent process, and is a common theme in many forms of acquired hearing loss. There is a suggestion that calcium channel blockers may reduce damage caused by apoptosis.

There is also some interesting evidence that the composition of intestinal microbiota may influence the expression of hearing impairment.

Some authors recommend dietary counselling be an integral component in the strategies to address hearing impairment.

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Meniere's disease is a subset of deafness and is partially managed by a very low salt diet. However, it seems likely to me that at some point it will be deemed a neurodegenerative disorder as there is an increasing number of commonalities between Menieres and neurodegenerative disorders, and consequently management will be changed from "just a low salt diet" to neurodegen management.

What actions will you initiate when you see someone whose diagnoses include deafness, will you -

- ensure you have a communication strategy to maximise communication and minimise a sense of diminishment of the person with deafness?
- review prescribed medications that may impact nutritional factors that

have been associated with hearing impairment?

- include pharmaconutrition impacts on hearing impairment in your report to colleagues?

Conclusions

Research into deafness in general is very limited as deafness impacts on health and productivity have been significantly underestimated.

The evidence is increasing that mal-nutrition contributes to the risk of hearing impairment, throughout life.

By default, prescribed medications that impact nutritional factors associated with hearing impairment are likely to further exacerbate that hearing impairment.

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 type="checkbox"/> | CVA | <input type="checkbox"/> | Enteral Feed | <input type="checkbox"/> | MS | <input type="checkbox"/> |
| Arthritis | <input checked="" type="checkbox"/> | CVD | <input type="checkbox"/> | Fall | <input type="checkbox"/> | Osteoporosis | <input type="checkbox"/> |
| Cancer | <input type="checkbox"/> | Dementia | <input checked="" type="checkbox"/> | Fracture | <input type="checkbox"/> | PD | <input type="checkbox"/> |
| CCF | <input type="checkbox"/> | Dentures | <input type="checkbox"/> | Frailty | <input type="checkbox"/> | Pressure Area | <input type="checkbox"/> |
| Chest Infection | <input type="checkbox"/> | Depression | <input checked="" type="checkbox"/> | Gout | <input type="checkbox"/> | Renal | <input type="checkbox"/> |
| COAD | <input type="checkbox"/> | DM Type 1 | <input type="checkbox"/> | Hypertension | <input type="checkbox"/> | Ulcer | <input type="checkbox"/> |
| Confusion | <input type="checkbox"/> | DM Type 2 | <input checked="" type="checkbox"/> | Incontinent | <input checked="" type="checkbox"/> | UTI | <input checked="" type="checkbox"/> |

Food Allergies: deafness, OCD

Other: somatoform disorder, hyperlipidaemia, bipolar

Medications That May Adversely Affect Nutritional Status

Drug	Vits/Mins Affected	Protein Binding > 90%	Nausea / Vomiting	Constipation / Diarrhoea	Weight	Appetite	Taste Change	Dry Mouth	Thirst	Salivation	Drooling	Dysphagia	Blood Sugar Level
COLOXYL WITH SENNA		<input type="checkbox"/>		D			<input type="checkbox"/>	<input type="checkbox"/>			<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
EPILIM	B12, B6, biotin, Ca, carnitine, D, folate, Mg, niacin, Zn	<input checked="" type="checkbox"/>	NV	CD	↑	↔	<input type="checkbox"/>	<input type="checkbox"/>			<input type="checkbox"/>	<input type="checkbox"/>	<input checked="" type="checkbox"/>
FEBRIDOL		<input type="checkbox"/>	NV	CD			<input type="checkbox"/>	<input type="checkbox"/>			<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

Extra Drugs:

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

No recent relevant available biochemistry. Advisable to check plasma proteins (albumin, total proteins) as markers of nutritional status. The plasma proteins are the primary transporters for one of the prescribed drugs and hypoproteinaemia may alter its effects.

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

Epilim decreases biotin and carnitine absorption, and decreases availability of folate and vitamin D.

Epilim decreases SVCT1 expression and function in the intestinal epithelia. This likely indicates a negative impact on vitamin C absorption and availability.

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

Epilim associated with increased risk of altered thyroid function and altered glycaemic control.

Concurrent ingestion of febridol (paracetamol) and iron resulted increased rate of iron absorption and decreased extent of drug absorption; the authors advise drug and iron to be administered at different times from each other.

Mr AAZ is a well-built, pale, frail man with thyroidy eyes and who was sitting in a fallout chair in the Day Room; he initially responded to my presence when I went to speak to him.

Since Mr AAZ is pale, advisable to check iron levels and if low then short term (90-120 days) intervention recommended.

If Mr AAZ has a low Hb and normal iron levels then it is likely he is low in biotin - biotin is important in 5 stages of Hb formation and status is likely to be compromised as epilim competitively inhibits biotin absorption.

Mr AAZ has thyroidy eyes therefore advisable to check thyroid function; epilim is associated with altered thyroid function.

Epilim decreases vitamin D availability therefore advisable to check vitamin D levels and if low then intervention recommended. Vitamin D is inversely associated with the inflammatory response markers such as CRP, TNF- α and IL-6 – elevated IL-6 is associated with disturbed sleep in those with bipolar disorder.

Mr AAZ's diagnoses include arthritis and therefore chronic pain - there is now evidence that chronic pain is associated with causation of dementia. Nutritional factors that may be useful to consider in pain management include -

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- vitamin D – currently prescribed epilim which decreases vitamin D status. Evidence indicates increasingly brittle pain control with decreasing vitamin D levels.
- vitamin C - pain increases the reactive substances (formerly Reactive Oxygen Species) within cells. Vitamin C is important in quenching reactive substances and if there is insufficient vitamin C then cell status becomes compromised and the cells typically die which also causes pain. Currently prescribed epilim which has been found to decrease vitamin C absorption. May be advisable to

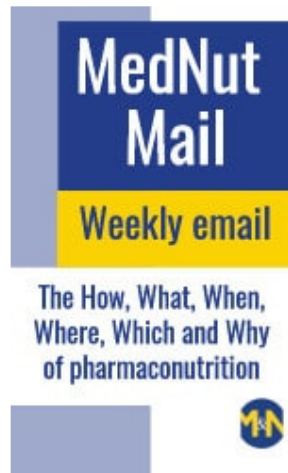
consider a vitamin C intervention - the optimal intervention is 500 mg vitamin C/day (if more than 500 mg vitamin C administered at a time then the excess above 500 mg is not absorbed as the vitamin C transporters are overloaded). Vitamin C is not considered part of the pain management armament however it won't cause harm and evidence suggests it may confer benefit.

What else would you include?

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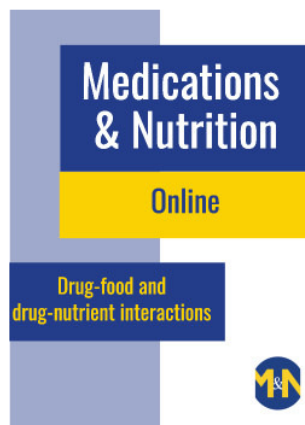
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