Multiple Sclerosis Journal
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Mult Scler 2013 19: 143
DOI: 10.1177/1352458513475916

The online version of this article can be found at:
http://msj.sagepub.com/content/19/2/143

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MULTIPLE SCLEROSIS JOURNAL | MSJ

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It is difficult to be the devil's advocate, the role of Caroline Papeix and Catherine Lubetzki, since the evidence, admittedly circumstantial and inferential, suggests that a) vitamin D in large doses is a potent immune modulator[1–4] and that a b) vitamin D deficiency is the major environmental factor in both susceptibility to, and severity of, multiple sclerosis. The studies of the prevalence gradient by latitude of multiple sclerosis and clinically isolated syndrome along east Australia, and in New Zealand are some of the many indirect sources of evidence to support this hypothesis.[5–7]

If the debate question was framed as *"Would I prescribe vitamin D 10,000 units to a patient with clinically isolated syndrome and an abnormal MRI scan?"*, then the answer is, of course, *"No"*. I agree with the antagonists in this debate that phase 3 trial data are needed before using vitamin D as a therapeutic agent. However, those trial data will take at least another 2-3 years to collect. Thus, while waiting for that proof, if I, or my children, developed a clinically isolated syndrome with an abnormal MRI scan, then I would have no hesitation in prescribing vitamin D in that dose for them.

A theme of previous commentaries has been a remarkable deficiency in clinician investigator-initiated trials in multiple sclerosis (the Scandinavians are notable exceptions). Why is this? Clearly we have no problem as neurologists participating in industry sponsored multicentre studies; we have the expertise both clinically and academically. ECTRIMS, ACTRIMS, LACTRIMS and PACTRIMS are healthy organizations, but they need to move to a level of endeavour and multi-national cooperation seen in the Oncology world. In addition we need the national multiple sclerosis societies to work together to fund such clinical investigator led studies. It certainly is impossible for any single national MS Society to fund a large phase 3 study needed to address this question. The distraction of CCSVI does not help.

Such a trial could recruit patients with a clinically isolated syndrome presentation and an abnormal MRI brain scan as the study population.[8] Even a relatively short term, 12 to 24 months, study, if adequately powered, could answer the question as to whether vitamin D in adequate doses (5000–10,000 IU daily) will prevent the development of multiple sclerosis. Hopefully the PREVANZ study, which has such a study population and intervention will answer the question in the next two years.[9] The Australian and New Zealand Multiple Sclerosis Societies and the neurologists in those two countries are to be congratulated in driving this through.

Some have questioned the safety of vitamin D. The case reported recently was taking supplementary calcium and was dehydrated; this was the reason for the hypercalcaemia.[10,11] Clearly in prescribing vitamin D one needs to take precautions in relation to conditions increasing susceptibility to hypercalcaemia (sarcoidosis, hyperparathyroidism, thiazide diuretics, et cetera) and the neurologist needs to screen for them.

Thus, while I accept the caveats from Paris, if one personalises the situation, I have no doubt that I would urge any relative, who does not have access to a clinical trial of vitamin D, to take vitamin D in the setting described.

Disclosures

Michael Hutchinson serves on a medical advisory board for the CONFIRM study [BG00012] for Biogen-Idec, serves on the editorial board of the Multiple Sclerosis journal, has received speaker's honoraria from Novartis, Biogen-Idec and Bayer-Schering and receives research support from Dystonia Ireland and the Health Research Board of Ireland.

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