Multiple Sclerosis (MS) is the most common immune-mediated inflammatory disease that attacks myelin and the axons of the central nervous system (CNS). MS can cause different cognitive and physical disabilities. The cause of MS is currently unknown, yet it is thought that there are many genetic and environmental components which effect MS. Some of the environmental risk factors include Epstein-Barr virus infections, cigarette smoking and low levels of vitamin D. It has been also shown that MS patients have a lower serum level of 25(OH)D (7,8). The prevalence of MS is directly related to latitude. The day length is shorter, colder temperatures, and less sunlight, which all attribute to less daily sunlight exposure in these areas. Therefore, these populations get less exposure to vitamin D in the United States, patients have a high incidence of MS. These observations give a strong reason to believe that there is a relationship between vitamin D and MS.

However, there is still uncertainty as to whether or not vitamin D and MS relapses and the possible mechanism by which vitamin D may be producing effects in MS patients. Relapse is defined as any worsening of clinical deficit such as weakness, paralysis, tingling, numbness, blurring of vision, ataxia, or balance problems which last for at least 24 hours occurring at least 30 days after the previous relapse. Some studies have shown that there is an inverse relationship between serum 25-hydroxyvitamin D levels and the rate of relapses in MS patients, as well as a predictor of MS activity and progression. Vitamin D supplementation has been shown to decrease the number of relapses and disease severity. It is thought that the relationship between vitamin D and MS is due to the effects that vitamin D has on the inflammatory response of the immune system, the immune response of immune cells and the activation of T-cells. It has been demonstrated that vitamin D inhibits CD4+ T cell proliferation, enhances IL-10 and inhibits IL-6 and IL-17, and induces both T-cell and regulatory T cells through an IL-22-mediated pathway. Still other studies have not shown these positive results with vitamin D supplementation, as well as other studies that are limited by a small sample size. More research needs to be continued to solidify the effects of vitamin D on MS patients. It is hypothesized that MS patients who are taking vitamin D will have a lower recurrence of relapse when compared to MS patients who are not taking vitamin D.

In this retrospective chart review, the relationship between vitamin D and MS relapse was investigated. The main results indicated that there is a non-statistically significant relation between vitamin D and whether or not patients had a relapse (p = 0.15). The results also indicated that there was no significant difference in the number of relapses for patients on vitamin D and on vitamin D. The study had negative results unlike previous studies such as the Pierrot-Deseilligny and colleagues study, which showed that 25(OH)D serum level related to a lower relapse rate in RNMD patients after vitamin D supplementation. However, our study looked at patients with all types of MS, not only RRMS patients. As previously stated, other MS types are characterized by a different relapse profile. The fact that all of our patients had another type of MS and in particular that 6.5% patients on vitamin D had SPMS, could have very likely skewed our data.

Disease modifying treatments (DMTs) were also not considered in our statistical analysis. The DMTs could have definitely had an impact on the number of relapses the patients had of the two year period. Studies have shown that vitamin D has a positive effect on MS progression. Ascherio and colleagues found that among patients with CIS randomized to early vs late treatment with IFN-β, with higher 25(OH)D levels had a lower degree of MS activity, MRI lesion load, brain atrophy and clinical progression during 5 years of follow-up. The CLIMB study showed that 25(OH)D had an effect on time to relapse and enhancing lesion load for MS patients on IFNβ and Glatiramer Acetate, however no effect on MS relapse. In our study, however, we did not know how the DMTs were started and stopped and the fact that patients changed DMTs within the interval of our study could not be considered.

It was also seen that 75% of the patients that were reviewed did not have any relapses. It is believed that having so many patients with zero relapses contributed to the fact that the distributions between vitamin D and non-vitamin D patients were not statistically different. This could have been due to a number of factors including patient follow-up, patients going to a different hospital at the time of their relapse, or patients failing to mention a relapse to their physician.

The uncertainty of the patient’s vitamin D compliance and levels could have skewed our results and misrepresented the vitamin D patient population. We also did not take into account whether patients on vitamin D were taking the proper dose. Rather we took into account whether or not vitamin D was mentioned in the chart during the time interval of interest. When the patients started vitamin D and how long they had been taking vitamin D could have also skewed our results.

The relationship between vitamin D and MS is an area of continued research. Much more research needs done in order to investigate this relationship further and the possible positive effect it could have on MS patients. This study helps us estimate the necessary sample size for a future prospective study adjusting for potential confounders. A future prospective study, where many more variables could be controlled and analyzed would be beneficial in investigating this relationship further.

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