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A CLINICAL REVIEW OF MICRONUTRIENTS IN HIV INFECTION

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ABSTRACT

The current research on the function of micronutrients in human immunodeficiency virus (HIV) infection is reviewed in this article. Nutritional deficiencies are prevalent in HIV-positive people. They develop as a result of malabsorption, abnormal metabolic, gut infections, and a weakened gut barrier. In HIV infection, there is a strong link between micronutrient shortages and immunological insufficiency, fast disease progression, and death. In addition, a vitamin A deficit increases the chance of vertical HIV transmission from mother to child, and a vitamin B12 deficiency increases the risk of neurological disability. Micronutrient research has been exciting in the past five years, and there is hope that certain micronutrients may be important contributors in preserving health and decreasing death in HIV immunodeficiency. Selenium seems to have a role in lowering HIV virulence and delaying disease progression. Vitamin A supplementation may decrease maternal mortality and enhance birth outcomes in HIV-positive pregnant women. Supplementation in HIV-positive youngsters may help them develop faster. Supplementing with carotenoids is being studied. Vitamin B12 may help to halt the development of HIV immune deficiency illness and restore neurological damage. In the context of a pre-existing deficit, the clinical effect of supplementing with certain minerals may be detectable. Apart from better overall diet, the effect of micronutrient supplements on health and the best way to utilize them in HIV infection is debatable due to the scarcity of controlled clinical studies. More study is required to better understand the function of micronutrient deficiencies in the progression of HIV infection, as well as the preventative and therapeutic significance of supplementing in HIV treatment. Nonetheless, recent evidence supports the use of regular multivitamin and mineral content supplements as a relatively low-cost adjunct to standard antiretroviral medication therapy.

KEYWORDS: *AIDS, HIV, Micronutrients, Trace Elements, Vitamins.*

1. INTRODUCTION

The significance of diet in human health has gotten a lot of attention in recent times. Vitamins supplement has been used effectively for a long time to treat and prevent a variety of clinical disorders. Vitamin A is used to maintain eyesight, beta-carotene is used to treat erythropoietin protoporphyria, vitamin C is used to treat scurvy, and niacin is used to treat pellagra, among other things. Numerous micronutrients have been found to have immunostimulatory and anti-

cancer effects in vitro and in animals, prompting several major epidemiological trials of micronutrient supplementation[1]. These studies, however, had no impact on the incidence or outcomes of cancer, stroke, or heart disease. Nonetheless, a protective effect of combination carotene, vitamin A, and selenium supplements on stroke risk was seen in a Chinese undernourished group[2].

Vitamin A supplementation has been shown to decrease morbidity and mortality in children suffering from infectious illnesses including measles, diarrhea, and acute respiratory infections. Significant decreases in the severity of diarrhea, respiratory illness, and malaria were seen in zinc supplementation studies. Selenium has been proven to defend against some malignancies, especially in the case of inadequate dietary intake[3].

Nutritional status assessment and corrections in HIV infection is becoming more widely acknowledged as an essential component of comprehensive HIV management. This article analyzes the function of supplementation with specific and combination micronutrients in HIV infection, evaluates the existing published literature on micronutrients in HIV infection, and makes recommendations for future study to further define the role of nutrition in HIV infection[4].

1. Micronutrients

1.1 Deficiency of micronutrients:

Micronutrient deficits are prevalent in HIV infection, and they may happen at any stage of the disease, including asymptomatic illness[5][6]. Serum levels of fat-soluble micronutrients and selenium are lower than other micronutrients, while serum carotene levels are lower than any other. There was no change in the frequency of micronutrient deficiencies in patients with CD4 cells more than 500, between 200 and 500, and less than 200 cells/L, according to Tomaka et al. Supplementing with micronutrients Among North America, multivitamin and trace element supplementation is prevalent in 63 percent to 73 percent of HIV-positive people. Despite the fact that multivitamin and trace element supplementation increased micronutrient levels at all phases of the illness, levels in HIV-positive patients were lower than in HIV-negative controls. Even with supplementation, 29 percent of HIV-positive people had one or more micronutrient deficiencies[7].Disease progression and mortality: Multivitamin use and intake of vitamin E, riboflavin, vitamin C, thiamine, and vitamin A were associated with slower disease progression in HIV. Tang et al observed slower progression of disease with moderate increase in intake of vitamins B1, B2, B6, and C, and reduced risk of mortality with all of these except vitamin C. Benefit was not significant with a great increase in intake of these micronutrients. These studies were observational in design, and residual confounding cannot be excluded as an explanation of results.

Birth outcomes: in HIV-positive mothers were investigated in a randomized, placebo-controlled study in Tanzania. In a factorial research design, the women were given vitamin A and/or multivitamins (vitamins B1, B2, B6, B12, C, and E, as well as niacin and folate, but not vitamin A). They were given ferrous sulphate and folate supplements on a regular basis, as well as preventive chloroquine once a week. Multivitamin supplementation was shown to reduce the chance of fetal loss by 39 percent, as well as the risk of low birth weight, severe preterm delivery, and small-for-dates birth by 40 percent[8]. There was also a substantial increase in

CD4, CD8, and CD3 cell counts. A modest therapeutic effect of vitamin A supplementation was seen, although it was not statistically significant[9].

1.2 Vitamin A:

Deficiency of vitamin Vitamin A insufficiency is prevalent at different phases of HIV infection, according to studies, even in 12 percent to 19 percent of asymptomatic HIV-positive people[10]. Levels seem to decrease as the illness develops, are more common in women than men, and may occur even when sufficient nourishment is provided. When compared to age-matched maternal controls, 63% of 474 HIV-positive pregnant women in Malawi had inadequate vitamin A levels, and 70% of their infants born with or without HIV infection had deficient vitamin A levels[11]. In one research, children were shown to have insufficiency before developing AIDS. Fat malabsorption, general malabsorption, diarrhea, gut infection, decreased gut barrier function, and altered metabolism all contribute to fatsoluble micronutrient deficits in HIV infection.

2. CD4 lymphocytes, disease progression and mortality:

Over an 18-month period, Baum et colleagues discovered a link between the development of vitamin A insufficiency and a substantial reduction in CD4 cell count. Low vitamins A levels were shown to be an independent predictor of mortality from AIDS-related causes. They also discovered that vitamin A insufficiency occurred in 20% of individuals who died from AIDS or infection during a 4-year period, while only 7% of HIV-positive control survived. 31 Serum retinol (vitamin A) levels were shown to be inversely related to the risk of death in HIV-infected intravenous drug users in another research.

3. Vertical HIV transmission:

A cross-sectional research found a strong negative association between prenatal vitamin A levels and viral load in breast milk of women with CD4 cell counts < 400 cells/mm3. Another research found a link between low vitamin A levels and increased viral shedding in vaginal secretions. Women who've been vitamin A deficient prenatal period were shown to be 3.69 considerably more probable to spread the virus to their offspring in an observational study of 133 HIV-infected mothers. With declining levels of vitamin A in the HIV-infected mother, there was a progressive increase in HIV transmission rates. A three- to four-fold increase in the probability of transmission was discovered using multiple regression. Infants born to mothers with the lowest levels of vitamin A perished within a year of birth. Vitamin A deficiency was linked to a greater chance of viral transmission, as well as having a dead or HIV-positive infant. Two observational studies involving 334 and 95 HIV positive expectant mothers found no link between low vitamin A levels and the likelihood of infected patients.

4. Vitamin A supplementation:

Viral load: In a small placebo-controlled trial of vitamin A and beta-carotene in HIV-infected pregnant women, no effect was seen on viral load. Similarly, no effect was seen on viral load with vitamin A and beta-carotene supplements in HIV-infected patients in two separate studies Vertical viral transmission, maternal mortality, and birth outcomes: Supplement vitamin A in expectant mothers at different stages of HIV infection had no overall impact on vertical viral transmission in randomized controlled studies. In a randomized, placebo-controlled study in 700 HIV-infected pregnant women in Malawi, prenatal supplementation with 10,000 IU of vitamin A

or placebo showed no impact on viral transmission at six weeks or 12 months. 47 In 750 women in South Africa, prenatal supplementation with 5,000 IU of vitamin A and 30 mg beta-carotene or placebo had no impact on virus infection, but there was a modest but statistically insignificant decrease in preterm deliveries. Mothers who took supplements were less likely to spread the virus to their preterm babies than women who took a placebo. Vitamin A and multivitamins (except vitamin A) were given prenatally in a large randomized, placebo-controlled study in Tanzania. In comparison to controls on placebo, there was no impact on vertical transmission in the prenatal or intrapartum periods, or for up to six weeks after nursing.

20,000 pregnant women in Nepal were given a weekly dosage of 23,300 IU vitamin A or beta-carotene in a placebo-controlled study. Both HIV-positive and HIV-negative women saw a 50% decrease in maternal mortality. Supplementation with the two micronutrients had no impact on outcomes related to birth weight, preterm, or newborn children in the same group.

I. Vitamin A supplementation in children:

Vitamin A was administered in single age-adjusted dosages to children of HIV-positive mothers in a randomized, placebo-controlled study in Durban at one and three months (50,000 IU), six and nine months (100,000 IU), and 12 and 15 months (100,000 IU) (200,000 IU). At 16 months, the treated group had 28 percent fewer episodes of diarrhea, 40 percent fewer bouts of diarrhea, and 77 percent fewer hospitalizations for diarrhea. Multivariate analysis revealed that the treatment effect was limited to children who tested HIV-positive. In a randomized, placebo-controlled trial from Tanzania, 687 children aged 6 to 60 months were hospitalized with pneumonia and given 200,000 IU vitamin A supplementation (half that if under 12 months old). The dosage was given again the following day, as well as at four and eight months. This resulted in a substantial increase in linear growth in HIV-positive children, ponderal growth in malaria-affected children, and stunting reduction in children with chronic diarrhea.

II. Oxidative stress and antioxidants:

Antioxidant imbalances in the host are linked to apoptosis, which may contribute to HIV development. 5 Reactive oxygen species (ROS) damage to lipid membranes, intracellular proteins, and DNA causes apoptosis. 17 By activating oxygen-responsive transcription factors, particularly NF-kB, ROS may awaken the latent HIV state, causing HIV replication in the infected Tlymphocyte. Antioxidant vitamins may help to prevent HIV replication and decrease ROS.

III. Safety of vitamin A:

Lengthy and large dose vitamins A usage has been linked to toxicity, as well as the possibility of HIV-1 expression.58 Toxicity appears as hypervitaminosis A and an increase in bone fractures.59 However, the risk of toxicity in the presence of HIV infection is unknown.

2. DISCUSSION

Because of the different research designs, dosages, durations of follow-up time, and study outcomes, the findings of micronutrient therapy studies are difficult to evaluate. Micronutrients levels in the blood are used to diagnose micronutrient deficits, however they may not accurately represent nutritional status. Gender, measurement time of day, acute illness, liver disease, technical characteristics, and recent consumption all affect micronutrients levels. There

may be interactions between micronutrients and concurrent antiretroviral medication treatment therapy, making extrapolation of results to other populations problematic. It's still uncertain if vitamin supplementation has any effect on the clinical course of HIV illness. Supplementation's therapeutic efficacy in various clinical situations and with different micronutrients requires further study. Nonetheless, there is substantial evidence that dietary deficiency has a negative impact on the course of HIV illness. Micronutrient supplements have also been proven to relieve symptoms, postpone the onset of AIDS, reduce mortality, accelerate child development, enhance birth outcomes, and reduce maternal mortality.

3. CONCLUSION

A number of interrelated variables affect the progression of HIV infection from the fusion of the initial virion with a CD4+ T-cell to AIDS and death. However, by better understanding the prognostic importance of a few of these factors, it may be feasible to enhance patient treatment and long-term outcomes. Although unchangeable, host variables are nonetheless significant in determining the patient's prognosis and directing therapeutic regimens. Furthermore, studying the interaction between the host and the virus has the potential to aid in the development of novel treatment methods. Immunological markers such as CD38 expression and the variety of HIV-specific cytotoxic lymphocyte responses provide information on the virus's antilogous control. Virological surveillance, particularly medication resistance supervision, will continue to play an important role in HIV infection management. Furthermore, as global access to antiretroviral treatment increases, the value of the need for low-cost, easily accessible disease indicators becomes clear. As with any disease of this size, a slew of variables must be considered in order to achieve the best possible quality of life and treatment outcomes.

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