

Original research article

Role of vitamin D in slowing the progression and treatment at pre-rheumatoid arthritis stage

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Abstract

A pre-rheumatoid joint pain stage, during which fundamental invulnerable go between (eg, autoantibodies and cytokines) can usually be detected, precedes the onset of clinically evident articular disease during the early rheumatoid arthritis phase. This normally advances into set up rheumatoid joint pain that is portrayed by constant aggravation and related tissue rebuilding and harm. Advances in explicit safe focused on therapeutics, including biologics and kinase inhibitors, have reformed clinical consideration and improved results astoundingly. There found no differences on 25(OH)D₃ serum levels between patients who later developed RA and healthy donors. A total of 58% of RA patients were not taking vitamin D supplements; the proportion of these with vitamin D deficiency (25(OH)D level <19 ng/ml) was 50%. This proportion was similar to that observed in control subjects (59.4%). One third of supplemented patients were still vitamin D deficient. In non-supplemented RA patients 25(OH)D levels were negatively correlated with the Health Assessment Questionnaire Disability Index.

Keywords: Vitamin D; Rheumatoid arthritis; 25(OH)D₃ and RA.

Introduction

Pre-rheumatoid joint inflammation (pre-RA) is utilized to assign event before the clinical event of RA. This stage is described by the presence of anomalies in invulnerable capacity and reactions without clinical indications of immune system tissue injury [1]. There is a preclinical period in the advancement of RA where the hereditary and ecological elements associate, presumably consecutively, to start and engender the immune system measure, bringing about tissue aggravation and injury. Also, illness related autoantibodies like rheumatoid factor (RF) and anti-citrullinated peptide antibodies (ACPA) can create without clinical signs and manifestations of tissue injury. At a later stage, negligible side effects or signs can create, which might be viewed as vague or unclassified for a specific rheumatic sickness prior to creating old style RA. Critically, all patients need not experience each phase of the illness, and the stages can be in a combinational way. For example, a patient can have hereditary danger variables, ACPA, and joint pain [2].

Rheumatoid joint inflammation is a chronic autoimmune disease that causes reformist articular obliteration and related comorbidities in vascular, metabolic, bone, and psychological domains. Rheumatoid joint pain influences about 1% of the populace, can introduce at whatever stage in life, and is more common in women than in men. The essential aetiopathogenesis is believed to be brought about auto immune dysfunction that developed in notional stages. A pre-rheumatoid joint pain stage, during which fundamental invulnerable go between (eg, autoantibodies and cytokines) can usually be detected, precedes the onset of

clinically evident articular disease during the early rheumatoid arthritis phase. This normally advances into set up rheumatoid joint pain that is portrayed by constant aggravation and related tissue rebuilding and harm. Advances in explicit safe focused on therapeutics, including biologics and kinase inhibitors, have reformed clinical consideration and improved results astoundingly. These medications have additionally disentangled the molecular and cellular nodes within the complex inflammatory networks that propagate and perpetuate the disease.

Vitamin D and rheumatoid arthritis:

It is clear that both genetic and environmental factors affect prevalence of autoimmune diseases. Therefore, the fact that vitamin D has been implicated as a factor in several different autoimmune diseases suggests that vitamin D might be one of the environmental factor that among others normally participates in the control of selftolerance [3]. The huge job of nutrient D mixtures as particular immunosuppressants is additionally delineated by their capacity to either forestall or notably stifle creature models of immune system illness [4]. RA is an immune system issue of multifactorial etiology in which both hereditary and nongenetic factors (for example irresistible, hormonal, ecological) add to illness defenselessness. Vit D may apply immunomodulatory impacts and hypovitaminosis D along with higher pervasiveness of RA appears to be regular among North when contrasted with South Europe [5]. As of late, more prominent admission of vit D was related with a lower hazard of rheumatoid joint pain (RA), just as lower nutrient D was found related with higher sickness movement [6,7]. In any case, different creators contend these outcomes that tracked down an opposite relationship between nutrient D and lower hazard of rheumatoid joint inflammation (RA), through polls to gauge dietary nutrient D admission [8]. These creators discovered no distinctions on 25(OH)D₃ serum levels between patients who later created RA and solid givers. There found no differences on 25(OH)D₃ serum levels between patients who later developed RA and healthy donors. To explain the contrasting conclusions, these authors suggested that direct measurement of vitamin D in serum is a more accurate estimate of vitamin D levels than is a dietary questionnaire, especially without taking sun exposure into account [6].

Vitamin D metabolism Endocrine pathway:

Vitamin D is produced after UV light stimulation of skin or is acquired from dietary sources, and is subsequently actuated in successive metabolic strides by explicit cytochrome P₄₅₀ compounds. In the first of these means, the compound nutrient D 25-hydroxylase, communicated fundamentally in the liver, produces the head circulating type of vitamin D, 25(OH)D. In the subsequent advance, 25(OH)D is utilized to the dynamic structure 1,25(OH)₂ D by 1 α -hydroxylase, which would then be able to apply natural consequences for target cells communicating the VDR. In this setting, flowing 1,25(OH)₂ D goes about as a chemical to keep up intestinal take-up of minerals—eminently calcium—and ideal bone capacity. While this endocrine model of nutrient D requires 25(OH)D for initiation by 1 α -hydroxylase, the viability of renal union of 1,25(OH)₂ D is additionally subject to other hormonal factors like parathyroid chemical (PTH) and fibroblast development factor 23 (FGF23 which positively and negatively regulate 1 α -hydroxylase activity, respectively [9].

The correlation between vitamin d and rheumatoid arthritis:

Several interventional studies have evaluated the relationship between vitamin D insufficiency and RA [10] researched the impact of nutrient D supplementation on RA, selecting two gatherings of patients: one with adequate nutrient D sum and one with nutrient D inadequacy. The last gathering was additionally partitioned into a subgroup accepting

nutrient D treatment (a-calcidol, 0.25 mg twice/day) and one under no pharmacological treatment. All gatherings were followed up for a very long time, and their visual simple scale (VAS), just as provocative biomarkers [C-receptive protein (CRP) and erythrocyte sedimentation rate (ESR)] and the quantity of excruciating and swollen joints were recorded each 2–3 months. No distinction was found between the nutrient D deficient subgroups. In any case, RA flare rate in the nutrient D adequate gathering was lower contrasted and the nutrient D-insufficient subgroup getting no treatment.

The commonness of nutrient D insufficiency was assessed in 1191 RA patients versus 1019 controls [11]. An aggregate of 55% of the RA patients were not taking supplementation and, inside this gathering, just 52% were nutrient D lacking, correspondingly to sound controls. Nonetheless, numerous RA patients who got supplementation actually remained nutrient D inadequate. Moreover, nutrient D conversely related with sickness movement and the Stein rocker useful state. When adjusting vitamin D levels for BMI and sun exposure time, the negative association between disease activity and vitamin D levels remained significant

Methods

Patients and controls

Present investigation is an imminent report led over a time of 18 months. The study population includes 300 consecutive patients (225 women, 75 men) The control group resulted from the merging of two population-based studies, representative of the general population recruited from osteoporosis centres equally distributed over the hospital. The principal study included 200 postmenopausal ladies matured 60 to 80 years not influenced by diseases or on therapy expected to change nutrient digestion [12]. The second group was comprised of 25 premenopausal healthy women 20 to 50 years [13]. The qualities of these two populaces were portrayed in subtleties somewhere else and the information dissected by the imminent report. The examination incorporates 300 successive RA patients (88% woman) and 75 controls, not on vitamin D enhancements, from laboratory. Along with boundaries of infection movement, useful impedance, and mean sun openness time, all patients had serum 25(OH)D estimated in a brought together imminent report

Results

A total of 58% of RA patients were not taking vitamin D supplements; the proportion of these with vitamin D deficiency (25(OH)D level <19 ng/ml) was 50%. This proportion was similar to that observed in control subjects (59.4%). One third of supplemented patients were still vitamin D deficient. In non-supplemented RA patients 25(OH)D levels were negatively correlated with the Health Assessment Questionnaire Disability Index, Disease Activity Score (DAS28), and Mobility Activities of daily living score. Significantly lower 25(OH)D values were found in patients not in disease remission or responding poorly to treatment, and with the highest Steinbrocker functional state. Body mass index (BMI) and sun exposure time were good predictors of 25(OH)D values ($P < 0.001$). The association between disease activity or functional scores and 25(OH)D levels remained statistically significant even after adjusting 25(OH)D levels for both BMI and sun exposure time.

Conclusions

In RA patients vitamin D deficiency is quite common, but similar to that found in control subjects; disease activity and disability scores are inversely related to 25(OH)D levels. When reduced the level of Vitamin D increased the level of 25(OH)D.

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