Oral Vitamin D Therapy

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Keywords: psoriasis ; oral vitamin D ; treatment

1. Introduction

Vitamin D is an essential nutrient in humans; it is produced by the body through exposure to the sun (the primary source of vitamin D), or more precisely, to mild ultraviolet B (UVB) light. Other sources of vitamin D include food and dietary supplements [1]. In 1928, the chemist and medical doctor Adolf Otto Reinhold Windaus was awarded the Nobel Prize for chemistry for the discovery of vitamin D [1][2][3]. Chemically, vitamin D_2 was first characterized in 1932, and vitamin D_3 was characterized in 1936. Currently, vitamin D is known as a hormone that regulates calcium-phosphorus homeostasis and protects the integrity of the skeletal system [4]. Vitamin D levels are influenced by many factors, including the season, period of sun exposure, time of the day, latitude, use of sunscreen, clothing, skin color, body weight, and medical conditions [5][6].

When epidermal cells are exposed to UVB, 7-dehydrocholesterol can be transformed into pre-vitamin D, which isomerizes to vitamin D_3 [7]. Next, vitamin D_3 undergoes 25-hydroxylation, through an enzymatic conversion in the liver, to form 25(OH) vitamin D (calcidiol), the primary circulating form of vitamin D. The plasma half-life of 25(OH) vitamin D is 2–3 weeks. Calcidiol is converted in the kidneys by 1-alpha-hydroxylation to the most active form, 1,25(OH)2D (calcitriol), which has a plasma half-life of 4–6 h [8]. This entire process is modulated by parathyroid hormone, hypophosphatemia, growth hormone, and other mediators.

Psoriasis is a chronic autoimmune skin disease with a strong genetic predisposition, characterized by sustained inflammation and followed by uncontrolled proliferation of keratinocytes and dysfunctional differentiation [9]. The first-line therapy for mild-to-moderate psoriasis is topical administration of corticosteroids and vitamin D analogues [10][11]. Keratinocytes and lymphocytes that infiltrate the lesions express the vitamin D receptor, which explains the effectiveness of this therapy in psoriasis [12].

The pathogenesis of psoriasis is not fully elucidated. The development of psoriasis plaques is mediated by Th1 cells and connected to keratinocyte hyperproliferation. This connection could explain the efficacy of immunosuppressive and antiproliferative vitamin D-like compounds, such as calcipotriol, in psoriasis [13]. Ligands for vitamin D receptor inhibit the expression of pro-inflammatory cytokines produced by T lymphocytes (i.e., IL-2, IFN-γ, IL-6, and IL-8) [14]. Thus, the biological activity of vitamin D_3 analogues leads to suppression of the T cell-mediated immune response. Moreover, dendritic antigen-presenting cells are modulated by 1a,25(OH)2D3 and its analogues, which inhibit the differentiation, maturation, activation, and survival of these cells [15]. Given current knowledge, it is reasonable to assume that epidermal production of vitamin D could be at least partially affected in skin psoriatic lesions, which may contribute to worsening symptoms.

2. Discussion

Although vitamin D has been used successfully for many years as a topical therapy in the fight against psoriasis, only recently have studies examined systemic vitamin D administration in psoriasis. We examined the pros and cons of this treatment, with the aim of determining whether systemic vitamin D would be a feasible therapeutic option for these patients. Among the existing reviews, very few were systematic in design. Indeed, from 1985 to the present, only a few studies have monitored the effectiveness of oral vitamin D in patients with psoriasis; consequently, the reviews were insufficient and inconclusive. Most studies did not observe side effects for doses within a relatively narrow range (0.25 to 2 µg/day). No evidence has been reported about the efficacy of the highest doses of systemic vitamin D in psoriasis.

However, most studies did not observe side effects. Based on these results, we can conclude that more large-scale studies are needed to determine the efficacy, optimal dosing, and adverse effects of vitamin D administration in patients with psoriasis.

References

1. Martens, P.J.; Gysemans, C.; Verstuyf, A.; Mathieu, A.C. Vitamin D's effect on immune function. Nutrients 2020, 12, 12 48. [Google Scholar] [CrossRef]
2. Windaus, A.; Linsert, O.; Luttringhaus, A.; Weidlich, G. Über das krystallisierte Vitamin D2. Ann. Chem. Liebigs 1932, 4 92, 226–241. [Google Scholar] [CrossRef]
3. Windaus, A.; Schenck, F.; von Werder, F. Über das antirachitisch wirksame bestrahlungs-produkt aus 7-dehydrocholest erin. Z Physiol. Chem. Hoppe Seylers 1936, 241, 100–103. [Google Scholar] [CrossRef]
4. Charoenngam, N.; Holick, M.F. Immunologic effects of vitamin D on human health and disease. Nutrients 2020, 12, 209 7. [Google Scholar] [CrossRef] [PubMed]
5. Brandão-Lima, P.N.; Santos, B.D.C.; Aguilera, C.M.; Freire, A.R.S.; Martins-Filho, P.R.S.; Pires, L.V. Vitamin D food forti fication and nutritional status in children: A systematic review of randomized controlled trials. Nutrients 2019, 11, 2766. [Google Scholar] [CrossRef] [PubMed]
6. Holick, M.F. Vitamin D deficiency. N. Engl. J. Med. 2007, 357, 266–281. [Google Scholar] [CrossRef]
7. Misra, M.; Pacaud, D.; Petryk, A.; Collett-Solberg, P.F.; Kappy, M. Drug and therapeutics committee of the Lawson Wilki ns pediatric endocrine society. Vitamin D deficiency in children and its management: Review of current knowledge and recommendations. Pediatrics 2008, 122, 398–417. [Google Scholar] [CrossRef]
8. Chang, S.W.; Lee, H.C. Vitamin u8h brD and health—The missing vitamin in humans. Pediatrics Neonatol. 2019, 60, 2 37–244. [Google Scholar] [CrossRef]
9. Rendon, A.; Schäkel, K. Psoriasis pathogenesis and treatment. Int. J. Mol. Sci. 2019, 20, 1475. [Google Scholar] [Cros sRef]
10. Slominski, A.; Kim, T.K.; Zmijewski, M.A.; Janjetovic, Z.; Li, W.; Chen, J.; Kusniatsova, E.I.; Semak, I.; Postlethwaite, A.; Miller, D.D.; et al. Novel vitamin D photoproducts and their precursors in the skin. Dermatoendocrinology 2013, 5, 7–19. [Google Scholar] [CrossRef]
11. Stanescu, A.M.A.; Grajdeanu, I.V.; Iancu, M.A.; Pantea Stoian, A.; Bratu, O.G.; Socea, B.; Socea, L.I.; Diaconu, C.C. C orrelation of oral vitamin D administration with the severity of psoriasis and the presence of metabolic syndrome. Rev. Chim. 2018, 69, 1668–1672. [Google Scholar] [CrossRef]
12. Burfield, L.; Burden, A.D. Psoriasis. J. R. Coll. Physicians Edinb. 2013, 43, 334–338. [Google Scholar] [CrossRef] [Pub Med]
13. Brown, A.; Slatopolsky, E. Vitamin D analogs: Therapeutic applications and mechanisms for selectivity. Mol. Asp. Med. 2008, 29, 433–452. [Google Scholar] [CrossRef] [PubMed]
14. Piotrowska, A.; Wierzbicka, J.; Żmijewski, M.A. Vitamin D in the skin physiology and pathology. Acta Biochim. Pol. 201 6, 63, 17–29. [Google Scholar] [CrossRef] [PubMed]
15. Penna, G.; Adorini, L. 1 Alpha,25-dihydroxyvitamin D3 inhibits differentiation, maturation, activation, and survival of den dritic cells leading to impaired alloreactive T cell activation. J. Immunol. 2000, 164, 2405–2411.