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+15878858911
editorial-office@sciformat.ca

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EXPLORING THE ASSOCIATION BETWEEN VITAMIN D AND URINARY TRACT INFECTION RISK - A REVIEW OF LITERATURE

Mateusz Banasik *(Corresponding Author, Email: mbanasik998@gmail.com)*
Collegium Medicum, University of Rzeszow, Poland
ORCID ID: 0000-0003-0256-8807

Aleksandra Adamczyk
Collegium Medicum, University of Rzeszow, Poland
ORCID ID: 0000-0001-7848-3506

Katarzyna Gunia
Samodzielny Publiczny Zakład Opieki Zdrowotnej Ministerstwa Spraw Wewnętrznych i Administracji w Katowicach im. sierżanta Grzegorza Załogi; Collegium Medicum, University of Rzeszow, Poland
ORCID ID: 0009-0003-0111-5758

Gabriela Grylowska
5 Wojskowy Szpital Kliniczny z Polikliniką w Krakowie, Poland
ORCID ID: 0000-0002-3796-2487

Anna Paluch
5 Wojskowy Szpital Kliniczny z Polikliniką w Krakowie, Poland
ORCID ID: 0009-0003-3913-0830

Marcin Rebizant
Szpital Uniwersytecki w Krakowie, Krakow, Lesser Poland, Poland
ORCID ID: 0009-0008-1159-228X

Agata Żak
Faculty of Medical Sciences in Katowice, Medical University of Silesia, Katowice, Poland
ORCID ID: 0009-0004-4520-9032

Andrzej Palak
Collegium Medicum, University of Rzeszow, Poland; Samodzielny Publiczny Zakład Opieki Zdrowotnej; Szpital Uniwersytecki w Krakowie, Poland
ORCID ID: 0009-0007-5381-8438

Anna Ignatowicz
5 Wojskowy Szpital Kliniczny z Polikliniką w Krakowie, Poland
ORCID ID: 0009-0006-6634-5564

Michał Tryba
Szpital Powiatowy w Chrzanowie, Poland
ORCID ID: 0009-0009-8363-5397

ABSTRACT

Urinary tract infection (UTI) represents one of the most prevalent bacterial infections worldwide. Emerging evidence suggests that vitamin D status may play a significant role in modulating susceptibility to UTI. This review article examined the available scientific literature concerning the association between serum 25-hydroxyvitamin D [25(OH)D] concentrations and the risk of UTI across diverse patient populations, including children, adults, pregnant women, and renal transplant recipients. The overwhelming majority of reviewed studies consistently demonstrated significantly lower serum 25(OH)D concentrations in patients with UTI compared to healthy controls. Serum 25(OH)D levels below 20 ng/mL were identified as a significant independent risk factor for UTI occurrence. The association was particularly pronounced in women and in the pediatric population.

The potential biological mechanisms underlying this relationship were also discussed, including the role of calcitriol in activating immune cells, stimulating the production of antimicrobial peptides - such as cathelicidin and defensins - modulating pro-inflammatory cytokine expression, and reinforcing epithelial barrier integrity. Functions and metabolism of vitamin D were reviewed, emphasizing its classical role in calcium homeostasis as well as its non-classical immunomodulatory actions. Vitamin D deficiency was identified as a modifiable risk factor that may be cost-effectively addressed through appropriate sun exposure and supplementation strategies. However, further well-designed, large-scale randomized controlled trials are warranted to establish causality and to determine whether correction of vitamin D deficiency can effectively prevent urinary tract infections.

KEYWORDS

UTI; Vitamin D; Vitamin D UTI; Vitamin D Immune System

CITATION

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Introduction:

Urinary tract infection (UTI) is one of the most prevalent bacterial diseases worldwide. According to a study by Yining He et al. (2025), the global disease burden of UTI demonstrated an overall upward trend between 1990 and 2021 [1]. A particularly pronounced disease burden was observed among elderly men, women, and regions characterized by a low Socio-demographic Index (SDI). However, considerable variation was noted with respect to sex, age group, country, region, and development level [1]. Global incidence is projected to continue rising until 2050, with an anticipated growth rate of 17.04% [1]. One of the preventive strategies against UTI is adequate nutritional status - encompassing sufficient intake of vitamins, minerals, proteins, and lipids - as a well-nourished organism is capable of mounting an effective defense against pathogenic microorganisms. The present review article analyzes the available scientific publications with regard to the relationship between vitamin D_3 status in patients and the risk of developing UTI.

Methodology:

The literature search was conducted using databases such as PubMed and Google Scholar.

Results:

The overwhelming majority of the available scientific literature suggests that low serum 25(OH)D concentrations are associated with an increased risk of UTI occurrence in adults, children, and pregnant women. However, further studies involving larger cohorts are warranted to confirm these observations and to evaluate whether correction of vitamin D deficiency may prevent urinary tract infections.

1. Functions of Vitamin D in the Human Body

The classical action of vitamin D involves the regulation of calcium homeostasis [2]. Vitamin D increases intestinal calcium absorption and stimulates calcium resorption from bone, while also supporting bone mineralization. It is supplied through dietary intake; however, relatively few food products constitute rich sources of vitamin D (>4 µg/100 g). These include, among others: numerous - though not all - fish species (5-25 µg/100 g), mushrooms (21.1-58.7 µg/100 g), reindeer lichen (87 µg/100 g), and fish liver oils (250 µg/100 g) [3]. Vitamin D may also be endogenously synthesized in the skin upon exposure to ultraviolet B (UVB) radiation, a process mediated by cutaneous melanin. Melanin absorbs UVB radiation and, through a cascade of photochemical reactions, an inactive precursor compound is formed, which subsequently undergoes hepatic hydroxylation to yield 25-hydroxyvitamin D_3 (25(OH)D_3). Although this form remains biologically inactive, it represents the most reliable indicator of overall vitamin D status in the body. Within the kidneys, this inactive form is further converted to the biologically active metabolite – calcitriol (1,25-dihydroxyvitamin D_3) [2].

Calcitriol exerts its effects by binding to the vitamin D receptor (VDR), forming a heterodimeric complex with the retinoid X receptor (RXR), which subsequently translocates to the cell nucleus and activates vitamin D-responsive genes. Numerous tissues beyond bone and intestinal epithelium also express the VDR - including bone marrow, the brain, the colon, breast tissue, neoplastic cells, and immune cells [4]. Antigen-presenting cells (macrophages and dendritic cells), as well as T and B lymphocytes, possess the capacity for both synthesis of and response to the active form of vitamin D, thereby enabling its localized immunomodulatory actions within the immune system [5].

Vitamin D was employed - albeit unknowingly - in the management of infectious diseases such as tuberculosis prior to the antibiotic era. Patients were referred to sanatoriums where heliotherapy (sunlight exposure therapy) was administered. Cod liver oil, a rich dietary source of vitamin D, was similarly utilized as an immunostimulatory agent [6]. Numerous observational studies have demonstrated an association between low serum vitamin D levels and an increased incidence of infections. A landmark study encompassing nearly 19,000 subjects demonstrated that individuals with serum 25(OH)D concentrations below 30 ng/mL reported upper respiratory tract infections more frequently. Analogous findings were reported in a study conducted among Finnish military recruits, in which those with lower vitamin D status exhibited higher rates of illness and increased absenteeism from duty [7].

2. Vitamin D and Urinary Tract Infections: Review of Current Evidence

Urinary tract infection (UTI) represents the most frequent bacterial infection encountered

in clinical practice [8]. Short-term complications of UTI can range from acute kidney injury and bacteremia to urosepsis and, in severe cases, death. In the long run, recurrent or chronic UTI may lead to proteinuria, elevated blood pressure, and progressive renal damage and failure [9]. Emerging evidence suggests that vitamin D plays a significant role in immune system functioning, which is of considerable relevance to the host response to UTI [10]. Several studies have investigated the association between serum 25(OH)D concentrations and UTI in women of reproductive age [11, 12], consistently demonstrating lower serum 25(OH)D levels in patients with UTI [12, 13].

In a study by Nseir W. et al. [13], 93 women with UTI meeting the study inclusion criteria were enrolled. Serum 25(OH)D concentrations were significantly lower in the study group compared to controls. The lowest recorded serum 25(OH)D level was 4 ng/mL, observed in a patient with recurrent UTI (at least six episodes per year), whereas the highest value of 46 ng/mL was recorded in a participant from the control group [13]. The authors concluded that vitamin D deficiency in premenopausal women was independently associated with recurrent urinary tract infections, while acknowledging that the precise mechanisms linking vitamin D deficiency to recurrent UTI remain incompletely elucidated [13].

A separate study examined the association between serum 25(OH)D concentrations and UTI in children, emphasizing that Escherichia coli is the predominant etiological agent of UTI in the pediatric population, accounting for 80-90% of infections [15]. This publication described a cross-sectional case-control study conducted in 101 Caucasian patients aged 1 month to 12 years. The study included 59 hospitalized patients

with UTI and 42 healthy controls. Participants with chronic conditions - including congenital heart defects, autoimmune disorders, and diabetes mellitus - were excluded from both groups.

Each enrolled patient received continuous vitamin D prophylaxis until the age of two years (400 IU/day during the warm season and 800 IU/day during the cold season), and 800 IU/day during the cold season after the age of two. The study demonstrated that serum vitamin D concentrations were significantly lower in children with UTI compared to healthy controls [14]. A higher prevalence of vitamin D insufficiency and deficiency was also observed among patients with UTI. Furthermore, the findings suggested that vitamin D may play an important role in preventing UTI recurrence, as evidenced by comparisons of vitamin D levels between patients with recurrent infections and those presenting with a first episode [14].

In a subsequent study by Tekin M. et al. (2015), the relationship between serum 25-hydroxyvitamin D_3 [25(OH)D_3] concentrations and the occurrence of UTI in children was investigated. According to the authors, vitamin D demonstrates potential antimicrobial activity against a broad range of microorganisms, including bacteria, viruses, and fungi [16]. Endogenously synthesized 1,25-dihydroxyvitamin D binds to vitamin D receptors, thereby stimulating defensin-encoding genes and promoting the synthesis of cathelicidin, a peptide with direct antimicrobial activity [17, 18]. The study enrolled 82 children aged 2 months to 12 years presenting with their first episode of UTI. Patients who did not fulfill the inclusion criteria were excluded prior to enrollment. Univariate analysis revealed a significant association between white blood cell count (WBC), serum C-reactive protein (CRP), and serum 25(OH)D_3 concentrations in the study group. Multivariate analysis demonstrated that a serum 25(OH)D_3 concentration below 20 ng/mL was significantly associated with UTI occurrence [16].

The authors concluded that vitamin D deficiency is associated with the development of urinary tract infections in children [16]. In a systematic review and meta-analysis conducted by Gan Y. et al. (2023) [20], the relationship between varying vitamin D levels and the likelihood of UTI in children was assessed. The analysis demonstrated that children with UTI had significantly lower serum vitamin D concentrations than healthy controls. Low vitamin D levels were significantly associated with UTI in children, and the likelihood of UTI was significantly increased when serum vitamin D concentrations fell below 20 ng/mL.

The authors concluded that serum vitamin D concentration, particularly when below 20 ng/mL, constitutes a risk factor for UTI in children [20].

In a case-control study by Chidambaram S. et al. (2022) [50], children aged one to five years were enrolled. Eighty-two children presenting with their first febrile UTI confirmed by urine culture constituted the study group, while 82 healthy children served as controls. The majority of UTIs in children are caused by Gram-negative enteric bacteria originating from fecal flora, which colonize the perineal region and ascend to the urinary tract. Escherichia coli is the most prevalent uropathogen and accounts for approximately 80% of pediatric UTI cases.

Other common pathogens include Klebsiella, Proteus, Enterobacter, and Enterococcus [51, 52]. In the study under discussion, E. coli was identified in urine cultures of 93% of the enrolled children. Serum 25-hydroxyvitamin D levels were measured in all participants; values below 30 ng/mL were classified as vitamin D deficiency. Mean serum 25-hydroxyvitamin D concentrations in the patient and control groups were 24.27 ± 9.70 ng/mL and 31.97 ± 10.7 ng/mL, respectively. Vitamin D deficiency was identified in 34 patients (41.5%) compared to only 10 children (2.2%) in the control group. The authors concluded that vitamin D deficiency may represent one of the risk factors for UTI in children, and that a significant association exists between vitamin D deficiency and febrile UTI in children aged one to five years [50].

In the prospective case-control study by Shalaby S. et al. (2018) [21], the primary objective was to investigate the association between serum 25-hydroxyvitamin D_3 [25(OH)D_3] concentrations and the risk of a first febrile UTI episode in children. The study enrolled 50 children presenting with their first febrile UTI without predisposing risk factors, along with 50 healthy age- and sex-matched siblings as controls. Complete blood count, serum C-reactive protein, calcium, phosphorus, alkaline phosphatase, and parathyroid hormone levels were assessed in all participants. Vitamin D status was determined by measuring plasma 25(OH)D_3 concentrations, with deficiency defined as a plasma level $\leq$25 nmol/L. Children with UTI exhibited significantly lower mean serum 25(OH)D_3 concentrations compared to controls. Patients with lower urinary tract infection had significantly higher serum 25(OH)D_3 levels than those with acute pyelonephritis. Mean serum 25(OH)D_3 concentrations were significantly lower in female patients compared to male patients, a difference not observed in the control group. Multivariate analysis demonstrated that serum 25(OH)D_3 $\leq$25 nmol/L was significantly associated with UTI. The authors concluded that vitamin D deficiency ($\leq$25 nmol/L) represented an independent risk factor for UTI in children [21].

In a case-control study by Seifollahi M. et al. (2024) [53], the roles of zinc and vitamin D as immunity markers in UTI were investigated in children without other predisposing risk factors. Zinc is required for the activation of vitamin D to enable it to fulfill its role within the immune system [54]. Vitamin D exerts systemic effects against pathogens and is essential for the regulation of immune responses. Hypocalcemia resulting from vitamin D deficiency may impair neutrophil function and lymphocyte activity. Among the key immunological functions of vitamin D is the inhibition of pro-inflammatory cytokines (IL-6, IL-8) and participation in limiting the inflammatory cascade activated following pathogen adhesion to the uroepithelium [55]. After excluding participants who did not meet the eligibility criteria, 40 patients were allocated to each of the study and control groups. The study group comprised 40 hospitalized children with UTI confirmed by positive urine culture. Patients receiving zinc and vitamin D supplementation, as well as those with comorbidities and known infection risk factors - such as diabetes mellitus, immunodeficiency, organ transplantation requiring immunosuppressive therapy, malnutrition, or severe sepsis - were excluded. Control group participants were recruited from children attending routine follow-up visits at the same hospital; only children without UTI symptoms, without zinc and vitamin D supplementation, without conditions affecting their metabolism, and with negative urine cultures were eligible. The study enrolled 80 children in total, comprising 52 girls (65%) and 28 boys (35%), divided into UTI and control groups. Results demonstrated significant differences between the two groups with respect to serum vitamin D and zinc concentrations [53]. Vitamin D deficiency was identified in 80% of children with UTI, compared to only 17.5% of healthy controls. Similarly, zinc deficiency was present in 60% of UTI cases, compared to 17.5% in the control group. The study demonstrated significantly lower serum zinc and vitamin D concentrations in children with UTI compared to healthy children.

The authors concluded that zinc and vitamin D deficiency may increase children's susceptibility to UTI, and that supplementation with these micronutrients may contribute to the prevention of recurrent infections [53].

The study by Mahmoudzadeh H. et al. (2020) [56] aimed to determine the association between serum 25-hydroxyvitamin D [25(OH)D] concentrations and UTI occurrence in children [56]. According to the authors, certain cross-sectional studies [57, 58] have demonstrated that serum vitamin D concentrations in children with UTI are lower than in healthy children, while a limited number of studies suggest that elevated vitamin D levels may represent a risk factor for UTI [59, 60]. The study compared serum 25(OH)D concentrations between 75 children with UTI and 75 healthy controls. Children eligible for inclusion were aged 2 to 7 years, presenting with their first UTI episode, manifesting symptoms such as fever, dysuria, abdominal pain, nausea, anorexia, and pyuria; having a positive urine culture; demonstrating normal nutritional status; having no renal disease; and having received no vitamin D supplementation within the preceding 12 months. Vitamin D deficiency was defined as a serum 25(OH)D concentration below 20 ng/mL. A significant negative correlation was identified between serum 25(OH)D concentrations and white blood cell count, neutrophil percentage, and disease duration. Normal vitamin D status (>30 ng/mL) was present in 27 (36%) children in the control group and in only 7 (9.3%) children with UTI. Mean (SD) serum 25(OH)D concentrations were 13.9 ng/mL in children with acute pyelonephritis and 18.7 ng/mL in children with cystitis. Vitamin D deficiency was diagnosed in 26 (81.25%) of 32 children with pyelonephritis and in 25 (58.1%) of 43 children with cystitis. The authors noted that the proportion of children with vitamin D deficiency was significantly higher in the UTI group than in the control group, corroborating an association between vitamin D deficiency and UTI. Furthermore, serum 25(OH)D concentrations were significantly lower in patients with acute pyelonephritis than in children with cystitis. The authors concluded that vitamin D deficiency was associated with UTI occurrence in children, and that the findings suggest vitamin D deficiency may constitute a risk factor for pediatric UTI [56].

In a case-control study by Haghdoost et al. (2019) [11], 187 participants were enrolled, comprising 97 pregnant women with confirmed symptomatic UTI (study group) and 90 healthy pregnant women (control group). Vitamin D deficiency (defined as serum 25(OH)D below 20 ng/mL) was demonstrated in 85.7% of women in the study group and in 52.2% of women in the control group. Serum 25(OH)D concentrations were significantly lower in pregnant women with UTI than in controls. The authors concluded that women with vitamin D deficiency are at increased risk of UTI during pregnancy [11].

In the study by Liu et al. (2022) [19], the objective was to investigate the association between UTI in adults and serum 25-hydroxyvitamin D (25(OH)D) concentrations, used as an indicator of vitamin D status. Serum 25(OH)D levels were retrospectively analyzed in 234 participants (190 females and 44 males), comprising 120 UTI patients (103 females) and 114 age- and sex-matched healthy controls (87 females).

Serum 25(OH)D concentrations were significantly lower in patients with UTI. Women with UTI exhibited significantly lower 25(OH)D concentrations than women without UTI. No significant association between serum 25(OH)D and UTI was identified in male participants. Multivariable logistic regression models demonstrated significant associations between UTI and serum 25(OH)D levels, female sex, neutrophil-to-lymphocyte ratio, and C-reactive protein. The authors drew two principal conclusions: firstly, that low serum 25(OH)D concentrations were associated with UTI; and secondly, that lower serum 25(OH)D levels in UTI patients were most pronounced among women [19]. No association was identified between serum 25(OH)D and diabetes mellitus, type of UTI (upper versus lower urinary tract), recurrent UTI, complicated UTI, or positive urine cultures [19].

The publication by Li X. et al. (2021) [22] presents a systematic review and meta-analysis aimed at evaluating the association between serum vitamin D concentrations and the risk of UTI in children. The authors report that the incidence of UTI varies from 1.8% to 7.5% in childhood [23], and that UTI is more common in boys under 3 months of age than in girls; however, girls are more likely to develop UTI after this age [24]. Studies reporting serum vitamin D levels in children with UTI and healthy controls were retrieved from PubMed, Scopus, Embase, and Cochrane databases. The results of the analysis demonstrated that serum vitamin D concentrations in children with UTI were significantly lower than those in healthy controls. Based on the findings, the authors concluded that a significant inverse association exists between serum vitamin D concentration and the risk of UTI in children [22].

In the study by Kwon Y.E. et al. (2015) [61], the impact of pre-transplant vitamin D deficiency on the development of urinary tract infection (UTI) following renal transplantation (RTx) was assessed. Vitamin D deficiency is frequently observed in renal transplant recipients, and given the indispensable role of vitamin D in immune system regulation, the authors hypothesized that an association might exist between vitamin D deficiency and post-transplant infections [61]. Serum 25-hydroxyvitamin D_3 [$25(OH)D_3$] concentrations were measured in 410 patients two weeks prior to RTx. Vitamin D deficiency was defined as a serum $25(OH)D_3$ concentration below 10 ng/mL. The primary endpoint was the occurrence of UTI following RTx. Mean serum $25(OH)D_3$ concentration was 12.8 ± 6.9 ng/mL, and 171 patients (41.7%) were classified as vitamin D deficient. Over a median follow-up period of 7.3 years, the incidence of UTI was significantly higher in patients with vitamin D deficiency (52 patients, 30.4%) compared to those without deficiency (40 patients, 16.7%). Statistical analyses confirmed that vitamin D deficiency was an independent predictor of post-transplant UTI [61].

3. Mechanisms of Action of Vitamin D

Based on the evidence presented in the aforementioned publications, a pertinent question arises as to why patients with low serum 25(OH)D concentrations - defined as below 20 ng/mL in most studies, though some publications applied thresholds of ≤25 nmol/L or even <10 ng/mL - exhibit a higher incidence of urinary tract infection (UTI). In a systematic review by Wimalawansa S.J. (2023) [25], the author demonstrated that vitamin D deficiency is associated with numerous chronic diseases and significantly increases the risk of infectious complications [26, 27, 28, 29]. Encouragingly, through appropriate public health guidance regarding safe sun exposure and supplementation strategies, vitamin D deficiency can be eliminated in a cost-effective manner, thereby reducing morbidity, premature mortality, and healthcare expenditure [25].

1,25-Dihydroxycholecalciferol (calciferol) represents the most biologically active metabolite of vitamin D and constitutes a potent immunomodulator essential for combating pathogens [30, 31]. The circulating hormonal form of calcitriol does not directly influence immune cell function; rather, the functional capacity of these cells depends on the adequate intracellular generation of calcitriol. Calcitriol activates cytosolic vitamin D receptors (VDR) following translocation to the cell nucleus, thereby modulating genomic functions, and additionally acts as a signaling molecule mediating non-genomic effects - including membrane-based effects as well as autocrine and paracrine signaling. The interaction of calcitriol with its receptor results in translocation of the receptor complex to the nucleus, where it binds to the genome and modulates the expression of over 1,200 genes [32]. Calcitriol attenuates inflammation and oxidative stress primarily through the suppression of pro-inflammatory cytokines. The immunomodulatory actions of vitamin D encompass the activation of immune cells - including T and B lymphocytes, macrophages, and dendritic cells - as well as enhanced production of antimicrobial peptides and neutralizing antibodies [33, 34, 35].

Chronic hypovitaminosis D also induces immune paresis, increasing susceptibility to infections - particularly intracellular bacteria such as Mycobacterium tuberculosis [36] and respiratory viruses [37, 38]. Furthermore, vulnerable individuals, such as elderly patients with comorbidities, demonstrate a strong

correlation between low vitamin D status and cytokine storm - a state of hyperinflammation driven by an uncontrolled, hyperreactive immune response [39]. Adequate vitamin D status reduces the risk not only of acute infections but also of chronic infectious diseases such as tuberculosis. Sufficient intracellular generation of calcitriol within immune cells regulates both innate and adaptive immunity, potentiating the innate immune response via monocyte and macrophage antimicrobial activity [40]. Calcitriol further modulates B lymphocytes and plasma cells to promote immunoglobulin synthesis and stabilizes B-cell homeostasis [41, 42], thereby augmenting antimicrobial peptide production. In addition to its effects on antimicrobial peptides, calcitriol induces autophagy and gap junction proteins [43] - forming tightly sealed intercellular junctions [44] - thereby reinforcing the structural integrity of epithelial and endothelial barriers and preventing viral penetration and paracellular fluid leakage [45]. Vitamin D also upregulates the expression of angiotensin-converting enzyme 2 (ACE-2) [46], suppressing the renin-angiotensin system and dampening inflammatory responses [47]. Increased expression of soluble ACE-2 neutralizes circulating viruses through direct binding interactions [48, 49].

All of the mechanisms described above may collectively contribute to a reduced risk of UTI development in individuals maintaining adequate serum 25(OH)D concentrations.

Discussion

The majority of the cited publications support the hypothesis that serum 25(OH)D concentrations are associated with urinary tract infection (UTI). Several studies indicate that low serum 25(OH)D levels increase the risk of UTI in pregnant women [11]. Another study demonstrated that combined supplementation with vitamin D and zinc may reduce the risk of recurrent UTI [56]. Similar conclusions were drawn in publications examining the association between serum 25(OH)D concentrations and UTI in the pediatric population [22, 56]. However, the reviewed literature lacks unequivocal evidence regarding the therapeutic potential of vitamin D supplementation in the prevention of UTI. Further well-designed clinical trials are needed to establish the therapeutic utility of vitamin D in UTI prophylaxis.

It is also noteworthy that a substantial proportion of the available studies focused exclusively on pediatric populations, whereas research involving geriatric patients remains notably scarce. Additional clinical investigations specifically targeting the geriatric population are therefore warranted.

Furthermore, there is a lack of conclusive scientific evidence suggesting that vitamin D administration demonstrates therapeutic efficacy in patients with an already established UTI. Patients diagnosed with UTI are treated with antimicrobial agents, and the discontinuation of antibiotic therapy for research purposes - with vitamin D administered as the sole intervention - would be both ethically unjustifiable and potentially harmful to patient health and safety. None of the reviewed studies specified the dosage of vitamin D required to achieve a clinically meaningful increase in serum 25(OH)D concentrations in affected patients.

Conclusions

The overwhelming majority of the available scientific literature suggests that serum 25(OH)D concentrations are associated with the risk of UTI occurrence in adults, children, and pregnant women. However, further studies involving larger cohorts are warranted to confirm these observations and to evaluate whether correction of vitamin D deficiency may effectively prevent urinary tract infections.

Authors' contribution

Conceptualization: Mateusz Banasik, Aleksandra Adamczyk, Agata Żak
Methodology: Anna Ignatowicz, Anna Paluch, Marcin Rebizant
Software: Michał Tryba, Aleksandra Adamczyk, Gabriela Grylowska
Formal analysis: Aleksandra Adamczyk, Agata Żak, Katarzyna Gunia,
Investigation: Andrzej Palak, Marcin Rebizant, Anna Ignatowicz
Resources: Gabriela Grylowska, Mateusz Banasik, Andrzej Palak
Data curation: Anna Paluch, Aleksandra Adamczyk, Mateusz Banasik
Writing - original draft: Aleksandra Adamczyk, Agata Żak,
Writing - review and editing: Agata Żak, Marcin Rebizant
Visualization: Agata Żak, Anna Paluch, Anna Ignatowicz
Project administration: Aleksandra Adamczyk, Mateusz Banasik

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