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EDITORIAL

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Dear Readers,
in this edition we continue to discuss about possible extra-skeletal effects of vitamin D, in completely different areas: dermatological and neurological.
There are two inflammatory skin diseases, namely psoriasis and atopic dermatitis, in which the alteration of the skin barrier seems to play an important role in their pathogenesis. The author, starting from the observation that psoriasis improves with exposure to sunlight and that the skin, irradiated by the sun, synthesises vitamin D, had previously conducted investigations into psoriasis, showing that vitamin D, in addition to its known functions, plays a role in the expression of some of the proteins constituting the Tight junctions (TJs), fundamental structures of the 'barrier organ'. Atopic dermatitis also improves with exposure to sunlight, and exposure to artificial sources of UV radiation is considered among the possible treatments for this dermatosis. Various hypotheses have been put forward to explain this phenomenon: immunomodulatory action of UV rays inducing apoptosis of inflammatory cells, inhibiting Langerhans cells and modifying cytokine production, or direct action of UV rays reducing Staphylococcus aureus colonisation, but the effect of sunlight exposure on vitamin D synthesis could also be considered. In this edition, the author summarises the results of his recent work [1] that brings new evidence on the relationship between vitamin D receptor polymorphisms, TJ protein expression and certain clinical manifestations in adult atopic dermatitis patients.
Vitamin D is known to be important for maintaining muscle strength through its action on specific receptors in muscle tissue. Patients undergoing rehabilitation, especially in the neurological field and in both inpatient and outpatient setting, are a high-risk population prone to developing a vitamin D deficiency and manifesting the consequences of this condition. In the second article in this edition, studies on the effectiveness of vitamin D supplementation during rehabilitation after stroke are considered. The author concludes that currently, the results are contradictory but that the available research has many limitations, including above all, as is often the case in other fields of study, the small sample size, the insufficient length of the observation period or the lack of preliminary assessment of vitamin status for which it cannot be excluded that non-deficient patients were included. By summarising the methods investigated and the results available to date, the article provides useful information for planning supplementation in the rehabilitation pathway of patients with ischaemic stroke, although further research is required to implement this knowledge in clinical practice.
In the recent OsMed report for the year 2023 of the Italian Medicines Agency (AIFA) [2], despite the prescription-based consumption [DDD/1000 inhabitants/day, Fig. (1)] and expenses of approximately 15% compared to the previous year, the expenses borne by the National Health Care Service for Vitamin D is of approximately EUR 200 million/year. It states that the data "confirm the use of cholecalciferol and metabolites for extra-skeletal indications for which RCTs have not provided evidence of efficacy". It also states that "the rich literature concerning the use of vitamin D in COVID-19 did not prove any benefit". For both of these statements, as can

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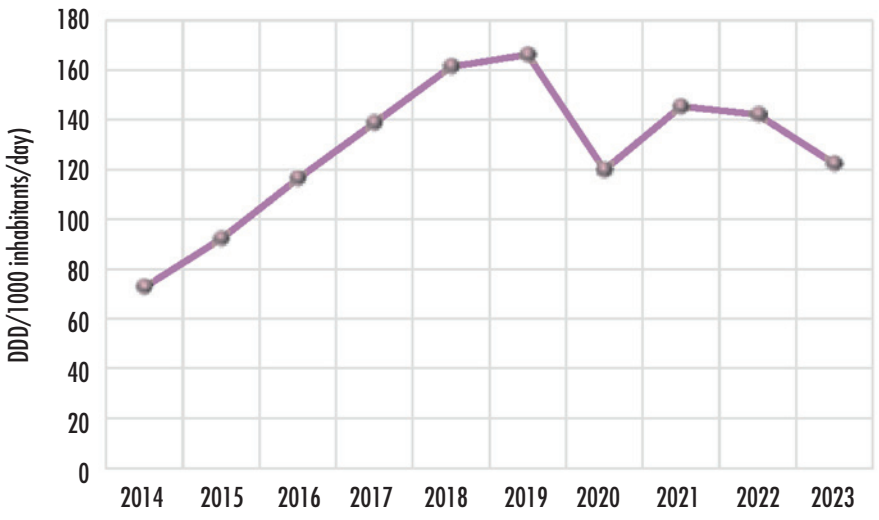

FIGURE 1.
Time trend 2014-2023 of vitamin D and analogs consumption (DDD/1000 inhabitants/day) (from AIFA, 2023, mod.) [2].

be seen from the bibliographic selection in our journal, it seems to me that the literature on the subject is in fact at least contradictory. In the same report I invite you to notice the curve in vitamin D consumption post note 96 in 2020, the first year of COVID-19, the recovery in the following years 2021 and 2022 and the subsequent decline in the post-COVID year 2023 (Fig. 1). In the above-mentioned OsMed report, it is also stated that vitamin D ranks third among the class A therapeutic categories purchased privately by the citizen with additional expenses of 76 million euro (26% of the total expenditure), an increase over the previous year.

What do you think?

Enjoy reading... and a Happy New Year

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2 Osservatorio Nazionale sull'impiego dei Medicinali. L'uso dei farmaci in Italia. Rapporto Nazionale Anno 2023. Roma: Agenzia Italiana del Farmaco 2024. https://www.aifa.gov.it/-/aifa-pubblica-il-rapporto-osmed-2023-l-uso-dei-farmaci-in-italia-

Atopic dermatitis and vitamin D

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By the term 'barrier organ' we mean the set of epithelia in the body, on whose integrity our survival depends. Numerous components contribute to the constitution of the barrier. In fact, there is a chemical, immunological, micro-biological and a physical barrier. The latter, at skin level, is much more complex than in other epithelia and is regulated by a well-defined set of molecules involved in the metabolism of filaggrin, the formation of the stratum corneum, the synthesis of intercellular lipid lamellae, the organisation of corneodesmosomes, desquamation and the formation of tight junctions (TJs), which reduce the intercellular spaces between epithelial cells until they disappear. In particular, it is the stratum corneum and the TJs, which are present in it, especially in the compact layer [1], that are responsible for permeability [2-5], which in other epithelia is provided by the TJ system. In addition, recent investigations have shown that partial or total inhibition of TJ proteins modifies epithelial permeability by interfering in the metabolism of filaggrin and lipids with abnormal formation of the stratum corneum [1,2].

In two inflammatory skin diseases, namely psoriasis and atopic dermatitis, barrier disruption appears to play an important role in their pathogenesis.

In fact, a series of investigations we conducted on psoriasis showed that vitamin D, in addition to its known functions, plays a role in the expression of some of the proteins making up the TJs. Starting from the observation that psoriasis improves with exposure to sunlight and that the skin, irradiated by the sun, synthesises vitamin D, we investigated the role of this vitamin. In particular, we have shown that in psoriasis patients, vitamin D is reduced and inversely correlates with PASI (Psoriasis Area Severity Index) [6] and with the blood level of T lymphocytes reg [7]; VDRs (Vitamin D Receptor) in psoriatic lesions are reduced by 50% compared to healthy skin; VDRs present polymorphisms that correlate with clinical [8]; finally, reduced VDRs are associated with reduced expression of certain TJ constituent proteins, in particular claudin-1, occludin and zonulin-1 [9]. In conclusion, in psoriasis, there is a correlation between vitamin D deficiency and altered TJ and, therefore, altered skin permeability. Based on these results, we turned our attention to atopic dermatitis (AD). Dermatosis is the cutaneous manifestation of atopy, a polygenic hereditary trait with high prevalence that can affect other epithelia (allergic rhinitis, asthma, etc.) in which, as already mentioned, the integrity of the barrier is also guaranteed by the TJ. It is more frequent in the paediatric age with a prevalence of between 10 and 30% and decidedly less frequent in adults (5-7%). It is an inflammatory disease characterised by altered permeability of the barrier due to filaggrin deficiency, the gene of which is mutated in 30-50% of patients. A Th2-type immunological response is predominant in AD with an increase in certain cytokines, such as IL-4, IL-5 and IL-13, which play an essential role in eosinophil recruitment and IgE synthesis. In this respect, it should be noted that two forms of AD are recognised: the extrinsic form, which is much more frequent and characterised by increased circulating IgE, and the intrinsic form, which accounts for approximately 20 % of cases, with normal IgE values. Finally, it must be remembered that AD is burdened with certain co-morbidities, such as psoriasis [10].

Moreover, AD also improves with exposure to sunlight [11]. In fact, exposure to artificial sources of UV radiation is considered among the possible treatments for this dermatosis [12]. The work of Napolitano et al. [13], in particular, shows that 6 out of 10 adults with AD improve after exposure to UV radiation. Various hypotheses have been put forward to explain this phenomenon: the immunomodulatory action of UV rays inducing apoptosis of inflammatory cells, inhibiting Langerhans cells and modifying cytokine production [14]; the direct action of UV rays reducing the colonisation of *Staphylococcus aureus*, but also the effect of sunlight exposure on vitamin D synthesis must be considered. In addition, studies have shown a correlation between severity of AD and the haematic concentration of vitamin D without being able to prove a link between these variables. Furthermore,

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different clinical trials on vitamin D supplementation in children have given contradictory results.

Therefore, in order to clarify a possible role of vitamin D, we enrolled 43 adults with moderate or severe AD who had not been on systemic therapy for at least three months and from who, after collecting anamnestic data, blood samples have been taken and skin biopsies have been done in the lesional (LS) and nonlesional (NLS) areas. In addition, EASI (Eczema Area Severity Index) was assessed for each of them and all of them have done the prick tests (STP) (Tab. I). PCR (Polymerase Chain Reaction) evaluations were carried out to study certain VDR polymorphisms (Tab. II) on blood, and histochemical examinations to study the expression of VDRs, occludin, claudin and ZO-1 on both NLS and LS and their gene expression. Multivariate logistic regression was used to explore the association between various VDR polymorphisms (dependent variables) or TJ proteins (dependent variables) and clinical and pathological characteristics of AD patients (independent variables). In this article, we will report the part of the results relating to the VDR [15] polymorphisms because not all of them have yet been published. The results of this cross-sectional study identify a link between VDR polymorphisms, VDR and TJ protein expression and clinical characteristics in a cohort of AD patients. Specifically, we observed a lower OR for the occurrence of AD in individuals with the hetero-zygote VDR polymorphism A1012G, whereas homozygous cumulative VDR polymorphisms ≥ 2 (Tab. III) were linked to an increased likelihood of developing allergic reactions. Among the constituent proteins of TJs, claudin and ZO-1 were those most expressed. VDR protein expression was associated with the presence of generalised AD lesions, whereas claudin showed a significant association with a positive SPT. While previous studies have investigated differences in the frequency of VDR polymorphism and the expression levels of VDR and TJ between AD patients and healthy control groups, the aim of our study was to characterise the interrelationships between SNPs (single-nucleotide polymorphism), VDR and TJ protein expression in skin biopsies from this cohort of AD patients and associate them with their clinical features. To date, no work has correlated VDR polymorphisms with the clinical characteristics

TABLE I.
Clinical and laboratory data.

Gender, no. (%)	
• Male	17 (39,5)
• Female	26 (60,5)
Age	
• < 60 years	36 (83,7)
• ≥ 60 years	7 (16,3)
Age of disease onset no. (%)	
• Childhood/adolescence	28 (65,1)
• Adults	15 (34,9)
Body Mass Index (BMI)	
• < 30 kg/m^2	35 (81,4)
• ≥ 30 kg/m^2	8 (18,6)
Location no. (%)	
• Flexure	9 (20,9)
• Generalised	18 (41,9)
• Head/neck	14 (32,6)
• Hands	2 (4,7)
EASI score	
• Mild (EASI < 16)	5 (11,6)
• Moderate-to-severe (EASI ≥ 16 or < 16 with involvement of the face and hands)	38 (88,4)
Asthma, no. (%)	
• Present	8 (18,6)
• None	35 (81,4)
Rhinoconjunctivitis, no. (%)	
• Present	14 (32,6)
• None	29 (67,4)
Skin prick test, no. (%)	
• Positive	20 (46,5)
• Negative	23 (53,5)
Total IgE (IU/ml), no. (%)	
• < 100 IU/ml	18 (41,9)
• ≥ 100 IU/ml	25 (58,1)
25(OH)D	
• ≥ 30 ng/ml	15 (34,9)
• < 30 ng/ml	28 (65,1)

of dermatosis. Our results suggest that VDR polymorphisms may indeed be associated with the clinical features of AD. We found that individuals with heterozygous A1012G status had significantly lower odds of developing AD early (OR: 0,046, IC 95%: 0.004-0.510, p = 0.012), suggesting a potential protective effect of this polymorphism on disease onset. Moreover, Richetta et al. reported a lower risk of developing psoriasis when this polymorphism, either in heterozygosity or homozygosity, was present compared to the wild-type gene [16].

We also observed that the presence of ApaI in homozygosity showed a tendency towards a higher probability (OR of 5.99) of developing the disease early, indicating a potential risk associated with this polymorphism. Interestingly, ApaI (rs7975232) is associated with lower levels of expression and reduced stability of mRNA VDR [17], and low levels of vitamin D are associated with AD. Moreover, Heine et al. [18] demonstrated an association between the ApaI polymorphism and severe forms of AD.

Our results showed a statistically significant association between the presence of homozygous cumulative polymorphism > 2 of the VDR and a positive SPT (10/20, 50%) compared to negative SPT (1/23, 4.3%; p = 0.0003). To support this observation, it has been reported that a low vitamin D level is associated with higher IgE levels in atopic patients [19,20].

VDR polymorphisms were also correlated with receptor expression in lesional skin biopsies (LS) from our cohort of AD patients. We observed a positive association for the ApaI polymorphism when in the heterozygous state with the expression of VDR. This result is in contrast to other results that report that polymorphisms in ApaI are associated with reduced messenger RNA stability and lower levels of expression [21,22]. However, another study in Turkish children described a significant link between ApaI in heterozygosity and risk of asthma [23]. It should be noted that functional studies on the association between ApaI polymorphisms and VDR protein expression are lacking.

With regard to the investigation of TJ constituent proteins, we observed that claudin and ZO-1 were the most highly expressed in skin biopsies of lesions from AD patients, whereas VDR and occludin were the lowest. No studies have reported the expression levels for these proteins. Only

TABLE II.
Target polymorphisms

Gene	Polymorphism	SNP ID	Position on chromosome 12 (assembly hg38)	Genomic location (NM 000376	ATG position in VDR (NM_000376)
VDR	A-1012G	rs4516045	47906043	VDR promoter	c.-1172A>G
VDR	FokI	rs2228570 rs107365810	47879112	Exon 3 (encoding)	c.2T>C
VDR	BsmI	rs1544410	47846052	Intron 9	c.1024+283G>T
VDR	ApaI	rs7975232	47845054	Intron 9	c.1025-49G>T
VDR	TaqI	rs731236	47844974	Exon 10 (encoding)	c.1056T>C

TABLE III.
Frequency of specific genotypes for different single nucleotide polymorphisms of the VDR in AD patients.

Polymorphism	Number of cases (%)
rs4516035 A1012G	
Genotype	N (%)
AA (wild type)	15 (34,9)
AG (heterozygous)	23 (53,5)
GG (homozygous)	5 (11,6)
rs2228570 FokI	
Genotype	N (%)
TT (wild type)	5 (11,6)
TC (heterozygous)	17 (39,5)
CC (homozygous)	21 (48,8)
rs1544410 BsmI	
Genotype	N (%)
GG (wild type)	18 (41,9)
GT (heterozygous)	20 (46,5)
TT (homozygous)	5 (11,6)
rs7975232 ApaI	
Genotype	N (%)
GG (wild type)	10 (23,3)
GT (heterozygous)	19 (44,2)
TT (homozygous)	14 (32,6)
rs731236 TaqI	
Genotype	N (%)
TT (wild type)	19 (44,2)
TC (heterozygous)	19 (44,2)
CC (homozygous)	5 (11,6)

case-control studies can be found in the literature. Furthermore, our work revealed a negative correlation between vitamin D and the expression of ZO-1 (rho = -0.43; p = 0.0058). In a study by Yuki et al. [24], TJ protein levels were quantified in the epidermal tissues of three AD patients and three normal subjects. Skin biopsies were taken from non-lesional sites of AD (NLS) and lesional skin sites (LS). In the NLS of DA, claudin-1, occludin and ZO-1 proteins detected conditions similar to those of normal skin. However, in LS, the signal intensities of claudin-1 and ZO-1 were markedly reduced. These data seem to be in contrast to our results, although only three patient samples were examined and our analysis was limited to LS.

Meckel et al. [25] observed a reversed correlation between serum 25(OH)D concentrations and mucosal inflammation in 230 subjects with ulcerative colitis, together with altered protein expression of VDR, occludin and decreased protein expression of ZO-1.

These results are in line with our results. We also found a positive association between ZO-1 expression and BMI (body mass index) ≥ 30. Zonulin is considered the only physiological mediator known to regulate intestinal permeability in a reversible manner by modulating intercellular TJs and obesity and has been associated with increased intestinal permeability and absorption [26].

We also observed a higher level of claudin expression in patients with positive SPT. De Benedetto et al. [27] reported reduced claudin-1 expression in DA NLS, while Gruber et al. [28] and Yuki et al. [24] showed that claudin-1 was over-regulated in the NLS of subjects with AD. Taken together, these results suggest a complex and context-dependent role of claudin-1 in AD, influenced by genetic factors and environmental considerations.

In conclusion, in our study of Italian AD patients, we identified significant associations between VDR polymorphisms, VDR expression, TJ proteins and clinical features of AD. These results provide important information on the complex interplay between genetic factors, vitamin D deficiency and TJ proteins in the pathology of AD, emphasising the complex nature of the pathophysiology of this dermatosis and the identification of potential markers for the early diagnosis of AD.

Finally, despite some similarities with psoriasis that we have highlighted, such as the protective role of heterozygous A1012G, the reduced expression of VDR, etc., differences emerge that, if correctly interpreted, could further clarify the role of vitamin D in the complex mechanisms regulating epithelial permeability.

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The role of vitamin D in post-stroke rehabilitation: between light and shadow

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Despite medical advances in the 20th century, vitamin D deficiency is still a pandemic 1: approximately 1 billion people worldwide suffer from vitamin D deficiency. Classically, vitamin D deficiency is associated with rickets in children while, in adults, vitamin D deficiency manifests itself as osteomalacia, a painful condition of defective skeletal mineralisation, or as osteoporosis causing skeletal fragility and fractures [1,2]. Vitamin D also plays an important role in regulating proliferation and differentiation in a variety of cells and tissues not associated with calcium metabolism [1-4]. Vitamin D receptors (VDRs) have been found in a variety of tissues and body cells, including brain, heart, breast, prostate, gonads, colon, pancreas, monocytes and activated T- and B lymphocytes [3-6]. Vitamin D is important for maintaining muscle strength through its action on VDRs in muscle tissue [7]. Inpatients and outpatients undergoing rehabilitation are a high-risk population prone to develop vitamin D deficiency and to manifest the consequences of this condition [8,9]. The functional results of these patients will depend on the correct diagnosis and appropriate treatment of the vitamin Deficiency.
In order to evaluate the effectiveness of vitamin D supplementation in patients undergoing rehabilitation programmes, we took into account studies concerning rehabilitation after cerebral strokes. The efficacy of rehabilitation after vitamin D supplementation in stroke patients was investigated, the efficacy of supplementation, and the type, form and amount of vitamin D administered. The inclusion criteria, the duration of the study and the scales used were discussed (Fig. 1).

THE EFFECT OF VITAMIN D SUPPLEMENTATION ON POST-STROKE REHABILITATION

Vitamin D deficiency may be associated with an increased risk of stroke onset, severity and future prognosis. It also affects cognitive decline and physical performance, which is observed in stroke patients who have worse outcomes in neurological rehabilitation [10].
Due to the numerous limitations of serum vitamin D testing and the method and quantity of its administration, there is little certainty as to whether rehabilitation outcomes in stroke survivors can be improved [11]. Many studies presented optimistic results and showed that vitamin D supplementation after stroke improved quality of life and facilitated patients' return to normal life.
Small sample studies showed better results for patients who supplemented with vitamin D after stroke [12,13].
Two small open trials conducted by Gupta et al. in India showed that intramuscular injection of high-dose cholecalciferol (600,000 IU) improved scores on various stroke scales and increased survival of post-stroke patients. A randomised, controlled, open trial included 73 patients with acute ischaemic stroke. Each patient was tested for serum 25(OH)D levels before entering the trial. A total of 53 patients with baseline 25(OH) D < 75 nmol/L were randomly assigned to the two trials. The first group received an intra-muscular injection of 600,000 IU of cholecalciferol once and oral cholecalciferol at a dose of 60,000 IU once a month with one gram of elemental calcium per day along with the usual post-stroke care. The second control group only received normal hospital care. Serum levels of vitamin D and iPTH were tested at 3 and 6 months after the study, and the follow-up itself lasted 6 months. The modified Rankin scale (mRS) was used in the study and high scores were obtained after 6 months. This result confirms the beneficial effect of vitamin D supplementation in post-stroke patients [10].
A randomised, controlled and unblinded study by Narasimhan and Balasubramani-

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Benefits of adequate vitamin D levels

More and more studies suggest that maintaining a normal level of vitamin D improves cardio- and cerebrovascular function. In addition, vitamin D influences the progression, development and prognosis of stroke.

HAEMORRHAGIC STROKE

- There is no scientific evidence that low vitamin D levels influence the risk of haemorrhagic stroke.
- Patients with haemorrhagic stroke often suffer from vitamin D deficiency and vitamin D supplementation reduces the damaging effects of the disease.

ISCHAEMIC STROKE

- Scientific studies suggest that low vitamin D levels may be associated with an increased risk of ischaemic stroke. $1,25(OH)_2D_3$ may induce the expression of IGF-, which contributes to the control and protection of nerve cells.
- IGF-1 has been shown to have anticoagulant properties via plasminogen activation.

FIGURE 1.
Role of vitamin D in ischaemic and haemorrhagic stroke.

an compared the results in patients with ischaemic stroke taking vitamin D supplementation with those without supplementation. The study used the Scandinavian Stroke Scale (SSS), a reliable and widely used scale in patients with ischaemic cerebrovascular disease. The first group of patients received a 60,000 IU dose of cholecalciferol administered by intramuscular injection; the second group did not receive vitamin D. Patients in both groups were examined at the beginning of the study and after three months. The results indicate a significant improvement in outcome among post-stroke patients taking vitamin D supplements [13]. Vitamin D supplementation in post-stroke patients had a positive effect on rehabilitation outcomes.

A small Japanese study by Momosaki et al. on 100 patients showed no improvement in stroke patients treated with vitamin D. After randomisation, each of the 100 subjects took an oral form of vitamin D_3 at a dose of 2000 IU/day or a placebo. Each patient received 450 capsules, each containing 400 IU of vitamin D_3, which means that the patients took the capsules five times a day. Vitamin D_3 was always taken at the same time, after lunch. The study with rehabilitation and vitamin D supplementation lasted eight weeks. During the patient's recovery and 8 weeks after discharge, the rehabilitation staff assessed each patient's Barthel index, Brunnstrom stage (arm, hand and leg on the affected side), hand grip strength (bilaterally) and calf circumference (bilaterally). A total of 97 patients completed the Japanese study. There was an improvement in Barthel index scores at week eight of rehabilitation as the primary endpoint; secondary outcomes were observed in Barthel index performance, hand compression strength and circumference of the calf. No differences were found in the other secondary endpoints between the groups.

None of these differences were statistically significant, indicating that daily supplementation with 2000 IU vitamin D_3 in patients after an acute stroke was ineffective and did not provide the expected benefits. A potential error was due to the fact that serum 25-hydroxyvitamin D levels were not considered as a patient inclusion criterion for the study; the researchers justified the choice with the assumption that almost all elderly patients undergoing rehabilitation have a vitamin D deficiency and, therefore, the deficiency study itself was not included in the patients participating in the study [14,15]. Other limitations highlighted by the authors include the small size of the study group, the short duration of supplementation and of the entire study, making it impossible to accurately determine the long-term and sporadic role of vitamin D supplementation in stroke patients. Another extremely important limitation was the comparison of a single vitamin D sample with a placebo sample [11].

The 2021 study by Torrisi et al. showed improvements in stroke patients undergoing deliberate neurorehabilitation and vitamin D supplementation, as well as in stroke patients undergoing vitamin D supplementation, after neurorehabilitation itself. A randomised, double-blind, parallel, single-centre, 12-week trial of 40 patients after ischaemic and haemorrhagic stroke was conducted. The participants were randomly allocated in a 1:1 ratio between two parallel groups: the experimental group in which 2000 IU/day of cholecalciferol was administered hourly and the control group in which the patients received no vitamin D supplementation. All patients enrolled in the study underwent intensive neurorehabilitation consisting of cognitive and motor training. All patients completed the rehabilitation cycle. All study participants were screened in two phases, at the beginning and at the end of the rehabilitation. The patients were assessed using the GSE scale, the Montgomery Aasberg Depression Rating Scale (MADRS) and the Functional Independence Measure (FIM). Serum levels of vitamin D and calcium were monitored. Significant improvements were observed in patients in the experimental

and control group in both psychological and functional performance. Patients taking vitamin D supplements showed greater variability than patients not taking them. The results indicated that intensive neurorehabilitation had a beneficial effect on functional recovery after a stroke; furthermore, a clear improvement was demonstrated in the experimental group, suggesting that vitamin D supplementation may also play a positive role [16]. However, vitamin D supplementation in stroke patients did not improve outcomes in a statistically significant manner.

According to Utkan Karasu and Kaymak Karataş, vitamin D supplementation can increase the effectiveness of rehabilitation in post-stroke patients. This is particularly important in patients who are in the first three months after a stroke and will undergo neurological rehabilitation for the first time. The retrospective study included 76 stroke patients. The patients in the study had a stroke (ischaemic/hemorrhagic) for the first time in their lives. The Brunnstrom Recovery Stage (BRS) for the lower limb and the Functional Assessment of Movement (FAC) scale were used to measure results in terms of motor function. Serum levels of 25(OH)D measured in ng/mg were examined during the first week of the study. The patients were divided into two groups: those undergoing vitamin D supplementation during rehabilitation and those who did not receive such supplementation. For 4-12 weeks, patients took oral vitamin D (50,000 IU) during rehabilitation and the total vitamin D dose ranged from 200,000 to 600,000 IU. Levels of vitamin D before rehabilitation and BRS and FAC scores, as well as changes in scores before and after the rehabilitation process, in stroke patients were recorded and compared in both the control and the study group. After the rehabilitation period, a positive and statistically significant change in FAC and BRS scores was found in the group receiving vitamin D. In addition, the effect of vitamin D supplementation on FAC and BRS scores in patients who started the rehabilitation treatment within the first three months after the stroke was compared. It was found that the change in FAC and BRS scores was statistically significant in patients treated with vitamin D. These results demonstrated the beneficial effect of taking vitamin D in patients during the rehabilitation after stroke. Vitamin D supplementation during post-stroke rehabilitation can have a positive effect on lower limb mobility and motor function according to Utkan Karasu and Kaymak Karataş [17]. In this study, vitamin D supplementation in stroke patients had a positive effect and patients had better rehabilitation results.

Sari et al. [12] investigated the effects of vitamin D supplementation on rehabilitation results and balance in patients with hemiplegia due to ischemic stroke. Seventy-two ischaemic stroke patients with low blood levels of vitamin D recovered in hospital for rehabilitation of hemiplegia were included in the study. A division into two groups was made: group A received vitamin D by intra-muscular injection (300,000 IU of vitamin D); group B received saline solution by intramuscular injection. Patients were examined at the beginning of the study and in the third month. To examine the effects, the Brunnstrom scale, the modified Barthel index, the Berg balance scale and the functional ambulation scale (FAS). By the end of the third month, a significant difference was found between the two groups in the modified Barthel index and Berg's balance scale. No statistically significant change was observed in the scores of the Brunnstrom scale or the Functional Ambulation Scale (FAS). In patients after ischaemic stroke, vitamin D supplementation (300,000 IU) did not significantly affect motor recovery and mobility. The study showed that vitamin D supplementation accelerated recovery and increased activity levels in patients. The result confirms the validity of the hypothesis that it would be appropriate to extend follow-up studies with more patients after stroke [12]. Vitamin D supplementation in post-stroke patients clearly did not improve outcomes.

A recent study was conducted in Poland on 94 patients undergoing rehabilitation treatment after an ischaemic stroke. The subjects included in the study (no. = 80) underwent a six-week rehabilitation therapy using proprioceptive neuromuscular facilitation (PNF, 60 minutes per day), mirror therapy (MT, 30 minutes per day) and occupational therapy (OT, 45 minutes per day). The Barthel index (BI) and the modified Rankin scale (mRS) were used for functional assessments. Laboratory tests were conducted for serum levels of vitamin D and Insulin-like Growth Factor-1 (IGF-1). There was a significant increase in BI scores (median difference = 2.0 points [pc]; $P < 0.001$) and IGF levels (median difference = 124.6 ng/ml; $P < 0.001$) after rehabilitation. There was a significant decrease in mRS scores (median difference = 7.0 pc; $P < 0.001$), but no significant difference in vitamin D levels ($P = 0.40$). The effect of age ($B = -0.01$, $P = 0.04$) and serum vitamin D level ($B = -0.02$, $P = 0.01$) on the BI score was demonstrated. The effect of body mass index (BMI) results ($B = -0.07$, $P = 0.02$) on the mRS score was observed. Lower serum vitamin D levels and older age may be associated with worse functional outcomes in patients with first ischemic stroke [18].

CONCLUSIONS

There is a lack of consistency in the results obtained among the studies investigating the correlation between supplementation with vitamin D in stroke patients and improved rehabilitation outcomes. Research has many limitations. Sometimes, serum 25-hydroxyvitamin D levels were not measured in the patients included in the study, which means that the authors selected patients with high or normal vitamin D levels rather than with deficiency. Another major limitation is the sample size, which is often too small. A larger number of participants might have led to different results, moreover, the authors considered different models, different administration regimens, quantities of vitamin D or even longer treatment times for patients. Often the short study period did not allow the long-term effects of supplementation to be examined. Since stroke is the leading cause of disability and the elderly often have severe vitamin D deficiencies, studies evaluating the effectiveness of vitamin D supplementation should be expanded. The results presented above include much information relevant to the planning of rehabilitation in ischaemic stroke patients in the recovery and compensation period, but further research is needed for the implementation of this knowledge in clinical practice.

There is increasing evidence that vitamin D has a positive impact on the prevention of cardiovascular diseases and contributes to better rehabilitation outcomes in stroke patients. Numerous studies testing the efficacy of vitamin D supplementation in post-stroke patients come up against the many limitations in methodology that invalidate the results. Due to the low number of studies and other limitations, it is not un-

equivocal that vitamin D supplementation in stroke patients always has a positive effect on improving rehabilitation. Considering that stroke is the leading cause of disability and that the elderly have high vitamin D deficiencies, it is necessary to expand studies testing the effectiveness of vitamin D supplementation. It is desirable that future studies on vitamin D supplementation in subjects undergoing rehabilitation treatment are controlled and randomised, conducted with a large sample of more than 1000 patients and with at least 5 years of follow-up.

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