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DETERMINE THE RELATIONSHIP OF VITAMIN D LEVELS WITH THE OCCURRENCE OF GRAFT VERSUS HOST DISEASE AS WELL AS INFECTIONS IN PEDIATRIC PATIENTS UNDERGOING HEMATOPOIETIC PROGENITOR CELL TRANSPLANTATION

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ABSTRACT

Vitamin D is known for its role in calcium homeostasis and is essential for bone health. It is now known to have other important functions in the body and its deficiency is associated with numerous pathological processes, both infectious and chronic degenerative. Recent studies have shown high prevalences (25 to 90%) of vitamin D insufficiency and deficiencyin sunny countries and locations, affecting all age groups. Allogeneic hematopoietic progenitor cell transplantation (HSCT) is important and potentially curative for hematologic malignancies, were graft-versus-host disease (GvHD) and infections affect outcomes. Vitamin D has emerged as a central player in immunity affecting T and B cells, macrophages and dendritic cells (DCs). A vitamin D-enriched medium maintains an immature DC corpus, associated with decreased ability to stimulate alloreactive T cells. Vitamin D deficiency has been associated with an increased incidence of chronic diseasessuch as graft-versus-host disease (GvHD), supplementation with vitamin D appears to induce an immunotolerance with DCs that has been suggested for the prevention of GvHD. One mechanism for this maintenance of immature DCs has been shown to be the positive regulation of indolamine 2,3-dioxygenase (IDO), an enzyme that transforms tryptophan to quinurenine which is a central feature in DC immunotolerance. Contrary to the suppressive effects on the immune system, vitamin D also has a protective effect against infections. This was from tuberculosis, where traditional treatments withsunlight and cod liver oil, rich in vitamin D, are replaced after the discovery of vitamin D-induced antimicrobial peptides. The two antimicrobial peptides under the influence of Vitamin D are LL-37 (cathelicidin) and β-defensin 2, which have activity against various bacteria, as well as against viruses and fungi.

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INTRODUCTION

Allogeneic hematopoietic stem cell transplantation represents the only possibility of cure for many patients with malignant and benign hematopoietic diseases. However, among its main complications are graft-versus-host disease (GVHD), as well as infections which limit its efficacy, being causes of morbidity and mortality after transplantation.

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The role of vitamin D and its effect on post-transplant complications have been evaluated in different studies, concentrating efforts in determining the immune modulation in GVHD and infections in transplanted patients.

Vitamin D is a fat-soluble vitamin that is indispensable in calcium homeostasis and bone metabolism.

The pathologic mechanisms of GVHD involve autoimmune and inflammatory processes. According to NIH guidelines, GVHD is classified as acute and chronic. Acute GVHD involves a reaction between donor alloreactive lymphocytes

and the tissues of the recipient's various organs, such as skin, intestine, and liver. This commonly occurs in 35-50% of patients presenting with intense inflammation, manifesting as diarrhea, rash, abdominal pain, cholestatic hepatitis, maculopapular erythema, usually occurring within the first 100 days post-transplantation.

The role of vitamin D in the immune system has been well defined in recent years, especially in autoimmunity and the adaptive immune response directed at host self-antigens [8 - 12]. Autoimmune disorders occur after a loss of self-tolerance and usually involve both lymphocytes and dendritic cells in their pathogenesis. Vitamin D receptorsare expressed on all cells of the immune system, and vitamin D has demonstrated effects on monocytes, dendritic cells, T lymphocytes and natural killer (NK) cells, both in vitro and in vivo [8, 13 - 15]. Vitamin D deficiency has been associated with several autoimmune disorders, including systemic lupuserythematosus (SLE), multiple sclerosis (MS), inflammatory bowel disease, rheumatoid arthritis, psoriasis, autoimmune prostatitis, and vitiligo vulgaris.

Vitamin D activity is measured by measuring 25-hydroxyvitamin D3, better known as calcidiol. Currently a level of 25-hydroxyvitamin D <20 ng/ml is considered as deficiency and levels of 20 and 29 ng/ml as insufficiency. Ranges between 30-60 ng/ml are considered optimal.

It is known that vitamin D has a very important role in immunomodulation, it has been demonstrated that it decreases the population of alloreactive lymphocytes (pro inflammatory), helping bone marrow recovery. Therefore, through these effects it generates benefits in transplanted patients.

In addition, it was demonstrated that vitamin D inhibits NK cells by decreasing the expression of CD69 and contributing to macrophage maturation, proliferation and maturation of B and T cells.

Vitamin D activity may decrease in transplant recipients as a result of decreased storage sites or production of vitamin D, prolonged lack of sun exposure, inadequate nutritional intake, decreased vitamin D absorption (due to gastrointestinal tract complications). These factors plus vitamin D deficiency may contribute to the development of GVHD.

It has been demonstrated that vitamin D deficiency is high, approximately 40% of the population and even higher in patients with hematological malignancies (48%). In patients undergoing hematopoietic progenitor cell transplantation it has been described that approximately 70% present vitamin D deficiency.

Some retrospective studies showed a higher incidence of chronic GVHD in patients who were vitamin D deficient before transplantation [8, 69].

Hansson et al. found that those with lower levels of 25 (OH) D3, pre-transplant, had lower survival with a higher relapse rate compared to patients with higher levels (36).

Particularly in engraftment, a study with adult post-transplant hematopoietic progenitor cells found that vitamin D supplementation improved bone marrow recovery 34% higher compared to control cells.

Kreutz et al. demonstrated that patients with grade III-IV GVHD had lower serum 25 hydroxyvitamin D3 levels than those with grade I-II [67]. This was also demonstrated in the study by Urbain et al. where low serum 25-hydroxyvitamin D3 levels after HCT were associated with moderate to severe GVHD [52].

MATERIAL AND METHODS

Pediatric patients aged 0 - 17 years undergoing hematopoietic progenitor cell transplantation at ABC Medical Center between January 2015 to December 2020, in whom 25-hydroxyvitamin D levels before transplantation were documented in the TIMSA electronic medical record.

A total of 100 patients were included patients were eligible for GVHD evaluation, For the analysis of infectious complications, only patients with follow-up in the Hospital were selected (n = 100).

The levels were measured in the clinical laboratory of the institution before transplantation, defining the values of deficiency and insufficiency according to the recommendations of the Pediatric Endocrinology Society.

In order to control the variables that influence the baseline status and, in turn, the outcome, patients with previous vitamin D supplementation or previous transplantation were excluded.

This is an observational, restrospective, cross-sectional, descriptive study.

Statistical analysis

Statistical analysis was performed using SPSS software. Summary measures were obtained for the dependent and independent variables with normal distribution. Analysis was performed with measures of association and impact. Median and interquartile range are shown. The probability obtained through the Wilcoxon rank sum test. Differences were considered statistically significant at P <0.05 bilaterally. Confidence intervals (CI) refer to 95% limits.

Table 1. Bivariate analysis of the variable Infection

Characteristic condition		No Infection	Infection	Total	P	OR (IC 95%)
Vitamin D						
	Normal	24 (38.71%)	11 (28.95%)	35 (35%)	-	1
	Deficiency	38 (61.29%)	27 (71.05%)	65 (65%)	0.323	1.55 (0.65 - 3.72)
Genere						
	Male	40 (64.52%)	23 (60.53%)	63 (63%)	-	1
	Female	22 (35.48%)	15 (39.47%)	37 (37%)	0.69	1.19 (0.51 - 2.74)

Age					
0 - 4	12 (19.35%)	4 (10.53%)	16 (16.00 %)	-	1
5 - 9	21 (33.87%)	7 (18.42%)	28 (28.00 %)	1	1
10 - 14	21 (33.87%)	18 (47.37%)	39 (39.00 %)	0.15	2.57 (0.68 - 9.75)
15 y +	8 (12.90%)	9 (23.68%)	17 (17.00 %)	0.106	3.38 (0.7 - 16.18)
Diagnosis					
Solid tumor	25 (40.32%)	5 (13.16%)	30 (30%)	-	1
H. Benigna	10 (16.13%)	3 (7.89%)	13 (13%)	0.624	1.5 (0.29 - 7.67)
H. Malignant	27 (43.55%)	30 (78.95%)	57 (57%)	**0.001**	5.56 (1.72 - 17.93)
Donor					
Autologue	32 (51.61%)	8 (21.05%)	40 (40%)	-	1
Allogeneic	17 (27.42%)	16 (42.11%)	33 (33%)	**0.01**	3.76 (1.26 - 11.2)
Haploidentic	12 (19.35%)	14 (36.84%)	26 (26%)	**0.005**	4.67 (1.44 - 15.13)
Umbillical Cordón	1 (1.61%)	0 (0%)	1 (1%)	0.623	-
Graft Days*	14 (12 - 15)	14.5 (13 - 18)	14 (12.5 - 15)	**0.01****	-
Cellularity*	5 (4 - 8)	6 (5 - 8)	5 (4.182 - 8)	0.18**	-

* Shown medium and RIQ
** Probability obtained through Wilcoxon's rank sum test

RESULTS

Our data showed an association between vitamin D deficiency and the presence of GVHD.

A secondary objective of this study is to establish the importance of supplementation before and during hematopoietic progenitor cell transplantation, since vitamin metabolism is largely impaired.

In this case, haploidentical transplantation was 14 times more likely to have infections compared to an autologous transplant.

This bivariate analysis indicates that vitamin D deficiency is

Table 2. Bivariate analysis of the GVHD variable

Condición característica	No EICH	EICH	Total	P	OR (IC 95%)
Vitamina D					
Normal	30 (46.88%)	5 (13.89%)	35 (35%)	-	1
Deficiency	34 (53.13%)	31 (86.11%)	65 (65%)	0.001	5.47 (1.76 - 17.01)
Genere					
Male	40 (62.5%)	23 (63.89%)	63 (63%)	-	1
Female	24 (37.5%)	13 (36.11%)	37 (37%)	0.891	0.94 (0.4 - 2.21)
Age					
0 - 4	14 (21.88%)	2 (5.56%)	16 (16.00 %)	-	1
5 - 9	17 (26.56%)	11 (30.56%)	28 (28.00 %)	0.064	4.53 (0.78 - 26.2)
10 - 14	22 (34.38%)	17 (47.22%)	39 (39.00 %)	0.029	5.41 (0.99 - 29.69)
15 y +	11 (17.19%)	6 (16.67%)	17 (17.00 %)	0.133	3.82 (0.58 - 25.02)
Diagnosis					
Solid Tumor	30 (46.88%)	0 (0%)	30 (30%)	NA	NA
H. Benigna	8 (12.5%)	5 (13.89%)	13 (13%)	-	1
H. Malignant	26 (40.63%)	31 (86.11%)	57 (57%)	0.303	1.91 (0.55 - 6.67)
Donor					
Autologue	38 (59.38%)	2 (5.56%)	40 (40%)	-	1
Allogenic	16 (25%)	17 (47.22%)	33 (33%)	< 0.001	20.19 (3.11 - 130.93)
Haploidéntic	9 (14.06%)	17 (47.22%)	26 (26%)	< 0.001	35.89 (4.09 - 315.15)
Umbillical Cordon	1 (1.56%)	0 (0%)	1 (1%)	0.821	-
Graft Days *	14 (12 - 15)	14 (13 - 16)	14 (12.5 - 15)	0.089**	-
Cellularity *	5 (3.5 - 7.94)	8 (5 - 8)	5 (4.182 - 8)	< 0.001**	-

* Shown medium and RIQ
** Probability obtained through Wilcoxon's rank sum test

not significant (p 0.323), compared to those who have normal vitamin D levels. Another significant variable is the days of grafting (<0.01), for the presence of infections. This is related to the fact that the longer/days of grafting, the longer the neutropenia time and with it the greater the risk of infections.

In this case, haploidentical transplantation had 17 times the chance of having GVHD compared to an autologous transplant. In the case of allogeneic transplantation, it was 17 times more likely to have GVHD compared to an autologous transplant.

This bivariate analysis indicates that vitamin D deficiency is also significant (p.001) compared to those with normal vitamin D levels.

Another significant variable is cellularity (<0.001). Since it does not exhibit normal behavior, we present it as a median and interquartile range (median 5). This means that patients with GVHD received higher cellularity.

This difference in the median between 8 and 5 is significant, demonstrating the high likelihood of an association between the variables presented.

The variables do not have free distribution, so they are presented as medians.

A significant association was shown with vitamin D deficiency and the presence of GVHD. The P value was significant (P < 0.01) for the association of vitamin D deficiency with GVHD.

In this case, patients undergoing haploidentical transplants were at higher risk of GVHD, as were patients undergoing allogeneic transplants, with P < 0.001 and P < 0.003, respectively.

In the case of infections, haploidentical transplants were three times more likely to develop infections compared to autologous transplants. Patients who received allogeneic transplants were statistically significant (p < 0.015).

This bivariate analysis indicates that vitamin D deficiency was not significant (p < 0.577) compared to those with normal vitamin D levels.

Another significant variable is the number of days of engraftment (0.005) for the presence of infections. This is related to the fact that the longer the days of engraftment, the longer the duration of neutropenia, and therefore the greater the risk of infections.

We can conclude that, for each day of engraftment, the risk of infection increases by 28%.

Table 3. Multivariate analysis for Infections and GVHD

Condición característica **OR (IC 95%)**	Infection		GVHD	
	P	OR (IC 95%)	P	
Vitamina D				
Normal	1	-	1	-
Deficiency	1.4 (0.43 - 4.52)	0.577	7.3 (1.61 - 33.11)	0.01
Genere				
Male	1	-	1	-
Femele	1.22 (0.43 - 3.47)	0.706	1.12 (0.31 - 3.99)	0.862
Age				
0 - 4	1	-	1	-
5 - 9	0.75 (0.1 - 5.69)	0.785	0.31 (0.02 - 6.31)	0.447
10 - 14	1.47 (0.22 - 9.64)	0.689	0.36 (0.02 - 6.39)	0.484
15 y +	1.12 (0.14 - 8.91)	0.913	0.19 (0.01 - 4.33)	0.298
Donor				
Autologue	1	-	1	-
Allogeneic	5.42 (1.4 - 21.04)	0.015	33.14 (3.29 - 333.52)	0.003
Haploidentic	3.34 (0.76 - 14.7)	0.11	138.96 (11.29 - 1710.72)	<0.001
Graft Days	1.28 (1.07 - 1.51)	0.005	0.95 (0.81 - 1.12)	0.566

In this case, the significant variable is the donor (<0.001), which is consistent with the clinical picture, since patients undergoing haploidentical or allogeneic transplants receive cells from different donors, which implies greater alloreactivity and triggers GVHD. This is also related to the cellularity required for each transplant, since a greater number of cells are administered in allogeneic and haploidentical patients. Similarly, vitamin D was found to be associated with the presence of GVHD.

Logistic regression with multivariate analysis was performed to determine the OR adjusted for the other variables.

None of the variables, such as age, donor, or sex, were associated with vitamin D deficiency and the presence of infections.

Vitamin D supplementation can enhance the effect of regulatory T cells and decrease the stimulation of cytotoxic T lymphocytes [70,72], increasing the immunomodulatory effect of vitamin D and thus preventing complications such as GVHD.

Engraftment is influenced by many factors, such as the source graft, cell dose, HLA mismatch, diagnosis, and conditioning regimen [87]. There is growing evidence that engraftment can improve with improved nutrition and vitamin supplementation.

D levels may be a surrogate marker of nutritional status [88]. Vitamin D promotes normal myeloid lineage differentiation and maturation and may therefore also play a role in engraftment [89].

DISCUSSION

Among its many effects, vitamin D has immunoregulatory properties. Among these properties, there is reasonable evidence that vitamin D and its deficiency may affect the course and outcome of HSCT. Despite a growing body of literature, the evidence is conflicting, and the effects of vitamin D deficiency on immune reconstitution and its impact on GVHD after HSCT remain poorly understood (98, 99).

Given its potential benefits, cost, and favorable side effect profile, consideration of vitamin D levels and supplementation could be easily incorporated into prospective GVHD studies, including clinical trials of novel therapies, supportive care, and biomarkers.

Our analysis confirms that patients with vitamin D insufficiency at the time of HSCT have a significantly increased risk of developing GVHD. Furthermore, our results also indicate that low pretransplant vitamin D levels do not represent a significant risk factor for infections, but are associated with graft failure and death.

This reinforces the argument that sufficient vitamin D levels during and after transplantation are important for establishing a healthy and balanced immune response.

We found no association between age, sex, and vitamin D levels, nor any association between vitamin D deficiency and infections, perhaps related to the sample size. Vitamin D levels and graft failure should be interpreted with caution due to the low incidence of this complication, and further studies are needed, particularly prospective, randomized studies of the effects of vitamin D.

Patients undergoing allogeneic HSCT, and especially those receiving haploidentical transplants, have been shown to have increased vitamin D deficiency (29,30). In line with this, we observed lower pretransplant vitamin D levels, which are exacerbated in the posttransplant period. An alternative is oral supplementation before or during immune reconstitution.

GVHD can be expected to reduce intestinal absorption of oral vitamins and vitamin D levels.

CONCLUSIONS

In summary, vitamin D deficiency may be associated with adverse outcomes after HSCT (GVHD, infections)

As reviewed, recipients of autologous and allogeneic transplants are at increased risk of vitamin D deficiency due to many factors, including underlying disease, treatment, and subsequent complications. Therefore, vitamin D deficiency is a modifiable risk factor for optimizing HSCT outcomes.

Currently, there are no established guidelines for vitamin D follow-up and treatment in transplant survivors.

Therefore, it is important to conduct future studies on vitamin D in HSCT to establish new guidelines.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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