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VITAMIN D DEFICIENCY IS ASSOCIATED WITH INCREASED FECAL INCONTINENCE SYMPTOMS

Candace Y. Parker-Autry, MD¹, Jonathan L. Gleason, MD², Russell L. Griffin, PhD³, Alayne Markland, DO, MSc⁴, and Holly E. Richter, PhD, MD⁵

¹Assistant Professor, Section on Urogynecology and Pelvic Reconstructive Surgery, Department of Obstetrics and Gynecology, Wake Forest University School of Medicine, Winston-Salem NC, USA

²Assistant Professor, Division of Female Pelvic Medicine and Reconstructive Surgery, Department of Obstetrics and Gynecology, Carilion Clinic, Roanoke, VA, USA

³Assistant Professor, Department of Epidemiology, University of Alabama at Birmingham, Birmingham, AL, USA

⁴Associate Professor, Department of Veterans Affairs (Birmingham VA Medical Center), Division of Gerontology, Geriatrics, and Palliative Care, Department of Medicine, University of Alabama at Birmingham, Birmingham, AL, USA

⁵Professor, Division of Urogynecology and Pelvic Reconstructive Surgery, Department of Obstetrics and Gynecology, University of Alabama at Birmingham, Birmingham AL, USA

Abstract

INTRODUCTION/HYPOTHESIS—Vitamin D is an important micronutrient in muscle function. We hypothesize that vitamin D deficiency may contribute to fecal incontinence symptoms by affecting the anal continence mechanism. Our goal is to characterize the association of vitamin D deficiency as a variable affecting fecal incontinence symptoms and its impact on health-related quality of life.

METHODS—This case-control study included women seen at a tertiary care referral center. Subjects were identified as having had a serum vitamin D level obtained within a year of their visit. Cases were women presenting for care for fecal incontinence symptoms. Controls were women without any pelvic floor symptoms presenting to the same clinical site for general

Corresponding author: Candace Y. Parker-Autry, MD, Department of Obstetrics and Gynecology, 4th floor Watlington Hall, Medical Center Boulevard, Winston-Salem, NC 27157, Phone: (336) 716-3779 Fax: (336) 716-4334 cparkera@wakehealth.edu.

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Author's participation:

Candace Parker-Autry – study design and implementation, data analysis, manuscript preparation

Jonathan Gleason - study design and implementation, data analysis, manuscript preparation

Russell Griffin - data analysis, manuscript preparation

Alayne Markland - study design and implementation, data analysis, manuscript preparation

Holly E. Richter - study design and implementation, data analysis, manuscript preparation

gynecology care. Cases completed the Modified Manchester Health Questionnaire and the Fecal Incontinence Severity Index to measure symptom severity and burden on quality of life.

RESULTS—Among the 31 cases and 81 controls, no demographic or medical differences existed between the groups. Women with fecal incontinence had lower vitamin D levels (mean 29.2 ± 12.3 cases vs. 35 ± 14.1 ng/ml controls respectively, $p=0.04$). The odds of vitamin D deficiency were higher in women with fecal incontinence compared to controls [OR 2.77, 95% CI (1.08–7.09)]. Among cases, women with deficient vitamin D (35%) had higher Modified Manchester Health Questionnaire scores, indicating greater fecal incontinence symptom burden [51.3 ± 29.3 (vitamin D deficient) vs. 30 ± 19.5 (vitamin D sufficient), $p=0.02$]. No differences were noted for fecal incontinence severity, $p=0.07$.

CONCLUSIONS—Vitamin D deficiency is prevalent in women with fecal incontinence and may contribute to patient symptom burden.

Keywords

vitamin D; pelvic floor; fecal incontinence

INTRODUCTION

Fecal incontinence is the involuntary leakage of solid or liquid stool and is the second most common pelvic floor disorder with an estimated prevalence of 9% in US women aged 20–59 years, increasing to 14.4% in women between 60–79 years.^{1,2} The anal continence mechanism involves primarily the proper function of the anal sphincter complex composed of the internal anal sphincter, the external anal sphincter, and the puborectalis muscles.³ While the etiology of fecal incontinence may be multi-factorial, weakness or disruption of any component of the anal sphincter muscle complex is a likely contributor.

Vitamin D is a fat-soluble micronutrient that plays a vital role in calcium homeostasis in smooth and skeletal muscle. In vitro studies suggest that serum vitamin D may impact skeletal muscle functional efficiency by regulating calcium homeostasis to affect muscle contractility, and by protecting muscle cellular environment against insulin resistance and inflammation. This relationship has been translated clinically as skeletal muscle efficiency and function improves with vitamin D supplementation in community dwelling women with insufficient serum vitamin D.

The Institute of Medicine recently concluded that a serum vitamin D level of ≥ 20 ng/ml was adequate for bone health. A normal serum vitamin D level is ≥ 30 ng/ml while levels < 29 ng/ml defines an insufficient vitamin D status.⁴ These definitions were created with respect to bone metabolism, and are extrapolated to non-skeletal conditions. Vitamin D insufficiency has become an epidemic in the United States; affecting 78% of geriatric adults and up to 80% of reproductive-age women.^{5,6} Vitamin D deficiency (serum vitamin D level < 20 ng/ml) is a more severe form of vitamin D insufficiency and is also a major public health issue with prevalence rates up to 39% in US adults aged 18 through 50 years.⁷

Vitamin D insufficiency/deficiency has received increased attention for its association with various extra-skeletal medical conditions^{8–12}; however, the most notable impact of

insufficient vitamin D is on musculoskeletal health. Vitamin D receptors are found in skeletal and smooth muscle such as the puborectalis and anal sphincter muscles that are essential in anal continence.¹³ Human in vitro studies consistently demonstrate that vitamin D regulates calcium homeostasis suggesting that serum vitamin D levels may have a role in smooth and striated muscle contraction, differentiation, and growth.^{8,14,15} Level I-II clinical studies have also supported the important role of vitamin D in the efficiency of muscle function.^{15–18} Together, these data support the biologic plausibility behind recent hypotheses that vitamin D has a role in optimal muscle health.

Insufficient/deficient vitamin D levels have been associated with increased colorectal and anal symptom distress in women with pelvic floor disorder symptoms including fecal incontinence.^{6,19} However, investigations into the relationship between fecal incontinence and deficient vitamin D levels are scarce. Our objective was to evaluate the role of vitamin D deficiency as a risk factor for fecal incontinence, to describe the prevalence of vitamin D deficiency and to characterize its relationship with fecal incontinence severity and anal sphincter function among women with fecal incontinence..

MATERIALS AND METHODS

Study population

Participants in this study were women seen in the Urogynecology Care Clinic at the University of Alabama at Birmingham between 2007 and 2011. All participants were identified by Current Procedural Terminology (CPT) codes to have had a total serum vitamin D level reported within 1 year of their evaluation in our clinic. Serum vitamin D panels were performed as a part of primary care screening independent of our practice. This study was approved by the Institutional Review Board at the University of Alabama at Birmingham.

All serum vitamin D panels were processed in the same outpatient laboratory using liquid chromatography.. [5, 7, 27] The 25(OH)D panel included total 25(OH)D (primary outcome measure) as well as 25(OH)D₂ and D₃ fractions.^{8,14,20} The same assay was used throughout the entire study period. Investigators performed a retrospective electronic medical record (EMR) review of women with vitamin D levels for inclusion and exclusion criteria. All subjects were women with at least one visit who were older than 19 years and had a serum vitamin D level reported within one year of their visit. The Urogynecology Care Clinic saw women for general gynecology care (controls) in addition to caring for women with pelvic floor conditions (cases). In order to specifically investigate women with fecal incontinence symptoms, women were excluded if they had pelvic floor symptoms without fecal incontinence symptoms or missing clinical data. To increase the external validity and generalizability of our data, women were excluded if they had any medical conditions known to impair absorption or metabolism of vitamin D, or to be a major cause fecal incontinence including: Stage 3 chronic kidney disease, chronic liver disease, gastric bypass, colovaginal fistula, and pelvic irradiation.

Variable definitions

For this case-control study, study groups were composed based on the presence or absence of fecal incontinence symptoms. Fecal incontinence was defined as the complaint of involuntary loss of solid or liquid stool according to the International Urogynecological Association and the International Continence Society definitions.² Cases were defined as women with fecal incontinence symptoms evaluated in our Genitorectal Disorders Clinic, a specialty clinic within the Urogynecology Care Clinic. All women seen at this clinic are referred by providers for evaluation and characterization of fecal incontinence. This evaluation includes completion of standardized questionnaires, anorectal manometry, and other testing as indicated. Controls were women without fecal incontinence or any other pelvic floor symptoms seen for general gynecology care.

The main exposure of interest, vitamin D deficiency, was defined as a total serum vitamin D level < 20 ng/ml. Women with total serum vitamin D levels ≥ 30 ng/ml were classified as vitamin D sufficient. Women with serum levels between 21–29 ng/ml were grouped as vitamin D insufficient. The total serum vitamin D level needed for general muscle health is yet to be defined, thus these definitions were extrapolated from bone health data.

Using a standard data form, the EMR was abstracted to obtain demographic, medical, and laboratory data. Demographic (i.e., age, height, weight, parity, and ethnicity) and medical characteristics were obtained from the initial history and physical examination documentation. Ethnicity was categorized as Non-Hispanic White or African-American. Body mass index (BMI) was calculated and reported as kg/m^2 . Medical characteristics abstracted for the case group included: (1) number of major medical conditions (obtained from past medical history), and (2) number of prescription/over-the-counter medications to include calcium and/or vitamin D supplementation taken regularly (obtained from the medication reconciliation documentation). In addition to the serum vitamin D level, laboratory data included a single value for the glomerular filtration rate (GFR) reported as $\text{ml}/\text{min}/1.73^2$. If more than one GFR was present, we used the reported value closest to the date of the vitamin D level.

Women in the case group completed the modified Manchester Health Questionnaire (MMHQ) and results were abstracted from the EMR to characterize fecal incontinence, its severity, and impact on quality of life. The MMHQ is a validated measure that includes the Fecal Incontinence Severity Index (FISI).²¹ The MMHQ measures health-related quality of life (HR-QoL) for fecal incontinence and includes eight subscales: overall impact, role, physical, social, relationships, emotion, sleep/energy, and severity/adaptation. The MMHQ total and subscale scores are scaled from 0 to 100 with higher scores representing greater impact on HR-QoL. The FISI component measured the severity of liquid, solid, mucus, or gas incontinence with severity scale varying from “2 or more times per day,” “once per day,” “2 or more times per week,” “once a week,” to “1–3 times per month.” Patient-weighted scores were used to determine severity of symptoms with scores ranging from 0 to 61, where higher scores indicate worse FI severity. A FISI score of 0 indicates continence.²¹ Anal manometry assessed anal sphincter function and anal sensation and was performed by a physician using a water-perfused disposable catheter system (Medtronic,

Inc, Minneapolis, MD). Pressures were recorded during resting, squeeze, and pushing at 2 cm from the anal verge. Rectal capacity was measured in milliliters using an air-filled balloon.

Statistical Analysis

The Student's t-test and Fisher's exact chi-square test were used for continuous and categorical variables, respectively, to compare demographic and medical condition characteristics between cases and controls. Unconditional logistic regression was used to calculate odds ratios (OR) and 95% confidence intervals (CIs) for the association between vitamin D deficiency and FI in the primary analyses. Two logistic models were created: one using dichotomous vitamin D deficiency as the dependent variable whereas women with insufficient serum vitamin D (serum vitamin D levels between 21–29 ng/ml) status were excluded in order to elicit the most significant clinical effect of vitamin D deficiency on fecal incontinence symptoms. The second model created used continuous serum vitamin D levels as the dependent variable. In a secondary analysis among cases, anorectal manometry pressures and MMHQ data were compared between cases that were and were not vitamin D deficient (serum vitamin D level ≥ 20 ng/ml) using the Wilcoxon rank-sum test. Analyses were performed using SAS v9.3 (Cary, NC).

RESULTS

Six-hundred and fifteen women were identified as patients evaluated in the Urogynecology Care Clinic who had a serum vitamin D level drawn within 1 year of their visit, and 374 were eligible for inclusion (Figure 1). Of these 374 eligible women, 44 met the case definition of having fecal incontinence and 103 met the control definition of not having any pelvic floor disorders. After excluding those with insufficient but not deficient vitamin D levels, there were 31 women in the case group and 81 women in the control group included in the primary analysis. There were no demographic or medical differences between these groups. The mean ($\pm$ SD) age and BMI of the group combined was 60.3 ± 1.2 years and 26.7 ± 0.3 kg/m², respectively; 78% were non-Hispanic White, 21% were African-American, and 53% used vitamin D supplementation. (Table 1) Over the past decade, many primary care physicians have routinely screened their patients for vitamin D nutritional status and supplement with vitamin D to achieve 'normal' levels in the absence of a targeted clinical outcome. In addition, vitamin D and calcium are encouraged for overall bone health in older women, therefore many women self-initiate supplementation. In our control population with sufficient vitamin D levels, 42/67 (63%) took vitamin D supplementation. Of those with deficient vitamin D levels, 4/14 (28%) took vitamin D supplementation. Among women with fecal incontinence (case group), of those with sufficient vitamin D, 9/20 (45%) took vitamin D supplementation. Similarly, 4/11 (36%) took supplementation of the group with deficient vitamin D.

Among women in the case group, 11 (35%) were vitamin D deficient and 20 (65%) were vitamin D sufficient. There were no demographic or medical differences between vitamin D deficient and sufficient women in the case group (data not shown). The mean age and BMI was 61.2 ± 0.1 years and 26.45 ± 0.2 kg/m², respectively for both groups. Twenty-five

(81%) were non-Hispanic white and 6 (19%) were African-American. The group mean parity was 2.3 ± 1.2 , ($p=0.10$). Forty-two percent used vitamin D supplementation.

Our primary analysis aimed to identify whether vitamin D deficiency was a variable associated with fecal incontinence symptoms. Case group serum vitamin D levels were found to be significantly lower than in the control group [$25(\text{OH})\text{D} = 29.18 \pm 12.25$ vs. $25(\text{OH})\text{D} = 34.97 \pm 14.07$ ng/ml, respectively; $p=0.04$] (Table 1) In addition, the odds of vitamin D deficiency was nearly 3-fold higher in women with fecal incontinence compared to controls [OR 2.77, 95% CI (1.08–7.09)].

Current definitions for vitamin D sufficiency and insufficiency are extrapolated from bone health standards, consequently we further explored the relationship with fecal incontinence by examining vitamin D levels continuously. In this analysis, participants with insufficient vitamin D levels were included in their respective group assignments as cases and controls. A total of 147 women (44 cases and 103 controls) were included in this analysis. As hypothesized, an inverse linear relationship was present between vitamin D serum levels and fecal incontinence symptoms where for every 5-unit decrease in total serum vitamin D level, there was a 19% increased odds of FI (OR 1.19, 95% CI 1.03–1.38).

To investigate the symptom impact of vitamin D deficiency on the quality of life of women with fecal incontinence symptoms (cases), we compared validated questionnaire data of women with sufficient vitamin D levels and deficient vitamin D levels. Modified Manchester Health Questionnaire data (Table 2) showed higher total and sub-scale specific scores in women with vitamin D deficiency. Women with vitamin D deficiency also sustained a greater negative impact from fecal incontinence symptoms, more social limitations, and a greater effect of fecal incontinence on sleep and energy compared to women with sufficient vitamin D levels. Fecal Incontinence Severity Index scores were also higher in women with deficient vitamin D levels compared to women with sufficient vitamin D levels, however, this difference did not reach statistical significance ($p=0.07$).

To explore a possible mechanism of how vitamin D deficiency may affect fecal incontinence symptoms in women, we compared diagnostic characteristics measured by anal manometry of women with fecal incontinence and vitamin D deficiency ($N=11$) to those with sufficient serum vitamin D levels ($N=20$). We found that the mean resting pressure at 2 cm was 33.7 ± 19.3 mmHg in vitamin D deficient women and 33.6 ± 13.7 mmHg in vitamin D sufficient women with fecal incontinence ($p=0.99$). Mean squeeze pressures at 2 cm was lower in vitamin D deficient women (60.7 ± 32.3 mmHg) compared to vitamin D sufficient women (73.9 ± 23.2 mmHg), but this difference did not achieve statistical significance ($p=0.21$). Vitamin D deficient women with fecal incontinence also had a lower mean change between resting and squeeze pressures at 2 cm (27.0 ± 22.1 mmHg) compared to vitamin D sufficient women with fecal incontinence (40.3 ± 21.2 mmHg), $p=0.12$.

DISCUSSION

In this care-seeking population of women with fecal incontinence, 35% were noted to have deficient vitamin D levels and had higher odds for having vitamin D deficiency in compared

to controls without fecal incontinence. In addition, the risk of fecal incontinence increased by 19% with decreasing levels of total serum vitamin D. Women with vitamin D deficiency and fecal incontinence also sustained a greater negative impact on health-related quality of life compared to women with sufficient vitamin D levels.

Our findings are supported by prior studies also suggesting that vitamin D deficiency is more prevalent in those with fecal incontinence in compared to the general population. Badalian and colleagues performed a cross-sectional analysis of the 2005–2006 cycle of the NHANES study. They found that among women older than 50 years with insufficient vitamin D levels, fecal incontinence was more prevalent compared to women with sufficient vitamin D levels [14.5%, 95%CI (12–17.4) vs. 12.5% (7.9–19.2), respectively. $p>0.05$].⁶ Additionally, Alkhatib and colleagues observed 10 cases of men and women with fecal incontinence, and similarly found that 60% of their cohort were vitamin D deficient.²²

Studies of the impact of vitamin D deficiency on fecal incontinence symptoms are also lacking. We previously reported findings of a separate retrospective cohort study in which we found that colorectal symptoms including fecal incontinence symptoms, were significantly more severe in women with vitamin D insufficiency compared to vitamin D sufficient women ($p=0.03$).¹⁹ Our prior data also align with our findings of higher total MMHQ and FISI scores in vitamin D deficient women with fecal incontinence.

Vitamin D has been shown to increase skeletal muscle cell proliferation and muscle fiber size in vitro,^{13,23} therefore it is biologically plausible for vitamin D serum levels to affect skeletal muscle efficiency through binding to the vitamin D receptor resulting in muscle growth.^{13,23} This hypothesis may explain a potential contribution to the mechanism behind our observation of increased odds of vitamin D deficiency in women with fecal incontinence symptoms compared to controls. Women with vitamin D deficiency may have less efficient use of the anal sphincter muscle complex thus potentially increasing the frequency and severity of fecal incontinence symptoms. The trends observed in the exploratory analysis of anal manometry squeeze pressures between vitamin D deficient and sufficient women with fecal incontinence are important hypothesis-generating findings that may support this theory. In addition to lower squeeze pressures among fecal incontinent women with deficient vitamin D levels, we observed a non-significant, but smaller mean change of anal sphincter pressure from rest to squeeze in comparison to women with sufficient vitamin levels. The clinical implications of this data are limited because the differences observed did not reach statistical significance likely due to our small sample. Thus, this data should be utilized as the basis of future research.

In light of these results, the current study should be viewed with certain strengths and limitations. First, the study is strengthened by the use of a control group to investigate the relationship between vitamin D nutritional status and fecal incontinence which extends the literature on this topic beyond case reports and cross-sectional studies. Our control group was of similar diverse ethnic diversity, geographic origin, age, body mass index, and number of medical co-morbidities as our case population, which strengthens the comparison between groups. This study is further strengthened by the robust data collected on the case

group to include validated measures of fecal incontinence symptom severity and impact on HRQoL and anal manometry which provides data regarding anal sphincter function.

The role of anal sphincter injury is important to consider. In our study, there were more women with anal sphincter defects seen on endoanal ultrasound among those with deficient vitamin D. It is plausible that the differences in impact of fecal incontinence on quality of life could be related to these differences. However, the differential of sphincter injuries between deficient and sufficient women with fecal incontinence did not reach statistical significance, therefore, these differences likely reflect chance. Subsequently, we did not control for sphincter injury in our regression analysis. The potential impact of deficient vitamin D nutritional status on fecal incontinence stands on the likely role in functional efficiency of the skeletal and smooth muscles of the anal sphincter complex. Further prospective exploration is needed to characterize the role of deficient vitamin D nutritional status and fecal incontinence symptoms.

More than 70% of women with fecal incontinence do not seek care. Therefore, this study is limited by the small sample size of the case group. Nevertheless, our sample size did allow for a 1 case to 2 controls comparison. It is plausible that women with fecal incontinence may have been included in the control group. However, because board-certified Urogynecologist were providing primary gynecologic care for the women included in the control group, it is likely that screening questions were included in the history and physical examination and if present, the fecal incontinence would have been treated. Generalized screening for vitamin D insufficiency or deficiency is not currently supported by evidence; therefore screening standards are variable among primary care providers which further limited our sample size. The potential of selection bias is inherent in the study design as women who present for evaluation may have more severe symptoms than those who did not present for care. This may potentially result in an overestimation or underestimation of the effect observed. The case-control design is also limited by the lack of definitive causality. Another limitation of the study was the lack of obstetric data in the control group as this data was not reliably reported in the electronic medical record as well as not accounting for stool consistency as a contributor to fecal incontinence.

CONCLUSION

This hypothesis-generating case-control study demonstrated that vitamin D deficiency was present in a significant proportion of women seeking treatment for fecal incontinence symptoms and had a negative impact on their quality of life. Prospective studies are warranted to further explore this relationship between vitamin D deficiency and more severe fecal incontinence symptoms. Specifically, further studies of anal physiology are needed to further our understanding of the impact of vitamin D on anal sphincter function. With further study, vitamin D supplementation may prove to be an important adjunctive therapy to pelvic floor exercises, biofeedback, anal sphincteroplasty, sacral neuromodulation, or pharmacologic therapy for fecal incontinence treatment.

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Figure 1.
Diagram of study population

Figure 2.
Graphical demonstration of the relationship between serum vitamin D levels and fecal incontinence symptoms.

Table 1

Demographic and clinical characteristics of cases and controls

	Cases (n=31)	Controls (n=81)	p-value [*]
Mean age, years	60.5±10.1	59.4±12.4	0.67
Ethnicity N (%)			
Non-Hispanic White	24 (80.0)	63 (78)	0.67
African-American	6 (30)	18 (22)	
Mean BMI	26.4 ±5.8	26.8±8.5	0.79
Mean number of medical problems	6.1±3.6	7.4±5.0	0.20
Mean number of medications	9.9±5.4	8.44±4.6	0.15
Vitamin D supplementation N (%)			
No	17 (56.7)	35 (43.2)	0.28
Yes	13 (43.3)	46 (56.8)	
Serum total 25(OH)D, ng/ml	29.18 ± 12.25	34.97 ± 14.07	0.04

* All data are based on t-test and Fischer's exact test for means and proportions, respectively and reported as mean ± SD or N (%)

Table 2

Questionnaire total and subscale scores of women with FI (cases), deficient and sufficient serum vitamin D

	Vitamin D deficient (N=11)	Vitamin D sufficient (N=20)	p-value [*]
MMHQ total score	51.3±29.3	30.0±19.5	0.03
MMHQ subsets			
Mean total FISI score	34.5±13.3	26.0±10.7	0.07
Incontinence Impact	68.2±40.5	36.1±40.4	0.05
Physical limitations	44.3±36.0	31.9±28.8	0.32
Social limitations	56.8±30.5	15.7±27.2	< 0.001
Sleep/Energy	42.0±35.4	17.4±21.9	0.03
Emotion	49.2±39.7	31.0±25.0	0.14
Global Health Perception	68.2±29.8	62.5±28.8	0.61
Personal relationships	30.7±36.8	18.8±30.1	0.35
Severity measures	50.9±31.5	39.7±28.0	0.33
External anal sphincter disrupted N (%), Yes	4 (40)	1 (6)	0.05

* All data are based on t-test and reported as mean±SD or N (%)