

Trichotillomania associated with a 25-hydroxy vitamin D deficiency: A case report

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Abstract

Vitamin D deficiency has been correlated with non-scarring alopecia including alopecia areata or female pattern hair loss. It was theorized that hair loss secondary to vitamin D deficiency in patients susceptible to trichotillomania may exacerbate this obsessive-compulsive disorder. Though vitamin D deficiency is common, especially among patients suffering from neuropsychiatric disorders, its correlation with trichotillomania is not well reported. Two female patients suffering from trichotillomania defined by noticeable hair loss on the scalp through the Massachusetts General Hospital Hair Pulling Scale were treated to promote hair growth. Treatment included dietary supplementation with vitamin D₃ 1000 IU every day. It was found that in both patients treated with vitamin D₃, marked improvements occurred over the span of 3 to 4 months. These included a reduction in obsessive compulsive disorder related hair loss as measured using the Massachusetts General Hospital Hair Pulling Scale, which correlated to their serum 25-hydroxyvitamin D levels. Experimental and clinical evidence is available to explain the underlying physiology and its probable relationship to trichotillomania's pathophysiology.

Keywords: vitamin D, hair loss, trichotillomania, obsessive compulsive disorders

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Background

Trichotillomania (TTM) is characterized by the *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition,¹ as

an obsessive compulsive and related disorder. The core symptoms of TTM include (1) recurrent pulling of one's hair, resulting in hair loss; (2) repeated attempts to decrease or stop hair pulling; and (3) clinically significant distress or impairment resulting from the hair pulling behavior.¹ Hair-pulling behavior may be precipitated by or occur concurrently with emotional states of anxiety, boredom, or as a result of tension.¹ Some individuals displaying hair-pulling behavior derive a sense of relief or pleasure from hair-pulling.¹ Hair-pulling may manifest as an automatic behavior in some individuals, while others may be more conscious of the hair-pulling. Most individuals report a mix of both behaviors.¹ Hair-pulling most commonly occurs from sites including the scalp, eyebrows, and eyelids, but may occur in any region of the body with hair.¹

Assessment of TTM is through use of the validated Massachusetts General Hospital (MGH) Hair Pulling Scale.² First described by O'Sullivan et al,² the MGH Hair Pulling Scale is a 7 question, self-rated scale that assesses



overall hair pulling tendencies by asking the patient to self-assess urges to pull, actual pulling frequency, perceived control over urges and actual pulling, and associated distress. The 7 questions are rated from 0 to 4 with a maximum overall score of 28, which indicates the greatest severity of TTM.

Trichotillomania has a 12-month prevalence of about 1 to 2% of the adolescent and adult population, with females being 10 times more likely to be affected than males.³ It typically onsets during or following puberty, around 10 to 13 years of age, and often occurs comorbid with other psychiatric conditions, with the most common being major depressive disorder and skin excoriation disorder.^{1,2} While the exact etiology of TTM is unknown, some familial studies have displayed the increased rates of TTM in first-degree relatives with the condition.³ If left untreated, TTM has a chronic course that waxes and wanes over time.¹

Empirical evidence exists for the use of habit reversal therapy for TTM where a patient tracks his or her hair pulling and undergoes awareness training, competing for response training, and stimulus control procedures that entails changing the environment to reduce triggers for hair-pulling.³ While no first-line treatment exists for TTM and a recent meta-analysis concluded that no particular medication class demonstrates efficacy in the treatment of TTM, anecdotal evidence suggests treatment effects of clomipramine, N-acetylcysteine, and olanzapine based on 3 individual trials with small sample sizes.⁴

Complex neuropsychiatric disorders such as TTM often lack pathophysiological or etiological explanations requiring clinicians to design therapeutic strategies in the absence of clear data. Therefore, treatment strategies are based on general causal information and short-term and long-term patient care considerations. Vitamin and mineral supplementation strategies not only assist with improved nutritional status of the patient but have been linked with preventing the development of a number of psychobehavioral paradigms. Specifically, vitamin D supplementation has been previously used to varying degrees in such disorders.⁵ Interestingly, aside from the possible hypothesized association with enhanced neuronal functioning, vitamin D is found in hair follicles, epidermal keratinocytes, and mesodermal dermal papilla cells, where it exerts its physiological influence through vitamin D receptors.⁶

Experimental evidence from cellular and animal models of hair disorders and clinical data suggest a role for vitamin D in regulating hair growth and development. For instance, in newborn patients with type 2A vitamin D-dependent rickets, alopecia developed within 1-3 months of age, and hair follicles displayed abnormalities such as dermal cysts

and irregular epidermal structures in the lower part of the follicle, confirming that vitamin D was essential for the hair follicle regrowth cycle.⁶ This work suggested a possible use for the inclusion of vitamin D in therapeutic regimens for the treatment of TTM, not only from the perspective of neuropsychiatric disorders but to promote sustained hair follicle regrowth following episodic damage.

Case Report

We describe 2 cases of TTM that presented to the pharmacy care clinic (PCC) for an initial assessment. Both patients had TTM onset during adolescence but had previously been successful in hiding the bald spots and had not received prior TTM treatment. The patients were not interested in psychotherapy, so treatment included only pharmacotherapy.

Case #1

The first patient was a 40-year-old white female with a past medical history significant only for vitamin D insufficiency and TTM. On the day of diagnosis of TTM, the patient was referred to and seen in the pharmacy care clinic. She was not taking any prescription or over the counter medications at this time. During the initial visit with the pharmacy care clinic, the patient scored 23 on the MGH Hair Pulling Scale. A picture of her mid-scalp area was taken, as seen in Figure 1A. During this visit, the patient was started on clomipramine 25 mg by mouth daily for treatment of TTM as well as vitamin D3 1000 IU by mouth daily. Her baseline labs, which included thyroid function (TSH with reflex T4) and general chemistry along with urinalysis were unremarkable. Her 25-hydroxy vitamin D level was 25 ng/mL (reference range 30-100 ng/mL). Over 5 months, the patient was seen 3 times for TTM management and completed the MGH Hair Pulling Scale at each visit. The clomipramine was not titrated and remained at a dose of 25 mg during the 5 months. Over the course of 3 visits, the patient demonstrated marked improvement in scores, specifically with a reduction in the urge to pull her hair. (Figure 1C) As seen in Figure 1A and B, she also had markedly improved hair regrowth in the mid-scalp area. Her 25-hydroxy vitamin D level at approximately 5 months after baseline visit was 33 ng/mL.

Case #2

The second patient was a 25-year-old white female with a history of anxiety disorder, and TTM first diagnosed. The patient presented to the pharmacy care clinic for baseline assessment of her TTM. A review of her

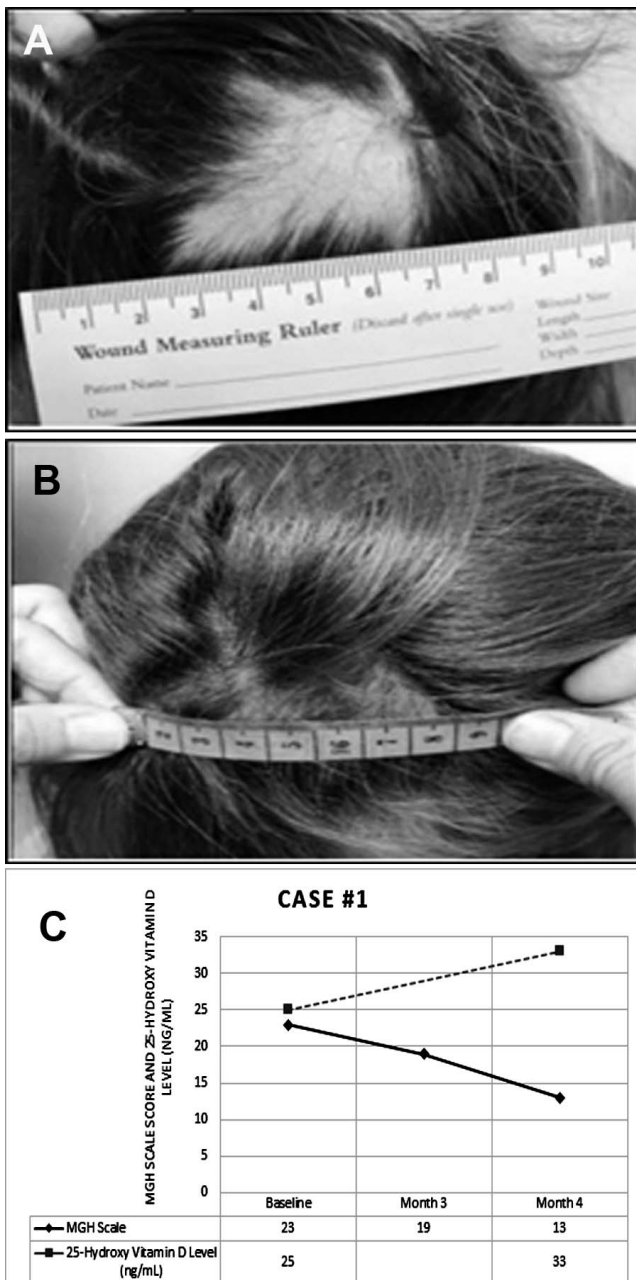


FIGURE 1: Before and after images of hair pattern and correlation analysis between hair pulling behavior and vitamin D supplementation for trichotillomania Patient One. (A) Shows the appearance of the hair in the first consultation, where a patch of hair loss was observed and measured in the mid-scalp region. (B) Depicts the hair distribution following treatment and during the fifth consultation. As shown, there is marked improvement in hair regrowth at the mid-scalp region after the patient was started on clomipramine 25 mg daily and vitamin D₃ 1000 IU daily. (C) Shows a correlation between the hair pulling behavior for Patient One, as defined and measured by the Massachusetts General Hospital Scale for Hair Pulling and serum levels for vitamin D.

prescription medications included only trazodone 50 mg by mouth daily. Her baseline labs included a thyroid function study, general chemistry, and urinalysis, all of which were unremarkable. Her 25-hydroxy vitamin D level was under 5 ng/mL indicating a severe vitamin D deficiency due to unknown cause. She was subsequently started on vitamin D₃ 1000 IU and clomipramine 25 mg by mouth daily. The patient was followed over the course of 3 months, and the MGH Hair-Pulling scale was administered at each visit. The clomipramine was not titrated and remained at a dose of 25 mg during the 3 months. Over the course of 3 visits, the patient demonstrated marked improvement based on MGH Hair-Pulling Scale scores, specifically with a reduction in the urge to pull hair (Figure 2C) and improved hair regrowth in the occipital ridge of the scalp (Figure 2A and B). Her 25-hydroxy vitamin D level increased to 32 ng/mL, 3 months after baseline visit.

Discussion

Antidepressants have long been used for the treatment of TTM, albeit with varying efficacy.⁷ Clomipramine was used for the treatment of the 2 cases presented here because of previously limited efficacy reported over other pharmacotherapy options. Though antidepressants have shown limited efficacy for TTM, hair regrowth is often not seen for many months following cessation of hair pulling.⁸ This case report identifies 2 patients with substantial hair regrowth and significant reductions in MGH scale scores, specifically with a reduction in the urge to pull, after only 3 to 4 months of treatment with clomipramine and vitamin D. It is thought that the clomipramine did not significantly contribute to reduction of TTM symptoms since both patients were not titrated to higher doses (150 to 250 mg) as is commonly done with TTM treatment. Though the second patient had a significantly reduced 25-hydroxy vitamin D level compared to the first patient, the decision to treat with the same dose of vitamin D₃ was due to newer evidence available in the literature since the 2009 vitamin D supplementation guidelines. A meta-analysis⁹ published in 2012 identified that vitamin D₂ and D₃ are equally efficacious at increasing serum 25(OH)D concentration.

Currently no documentation of vitamin D deficiency-associated TTM exists in the medical literature, though vitamin D deficiency has been linked to several chronic psychiatric illnesses including depression, obsessive compulsive disorder (OCD), and schizophrenia.¹⁰ Evidence suggests vitamin D likely has significant functions in the human brain with vitamin D receptors found in both neuronal and glial cells in the central nervous

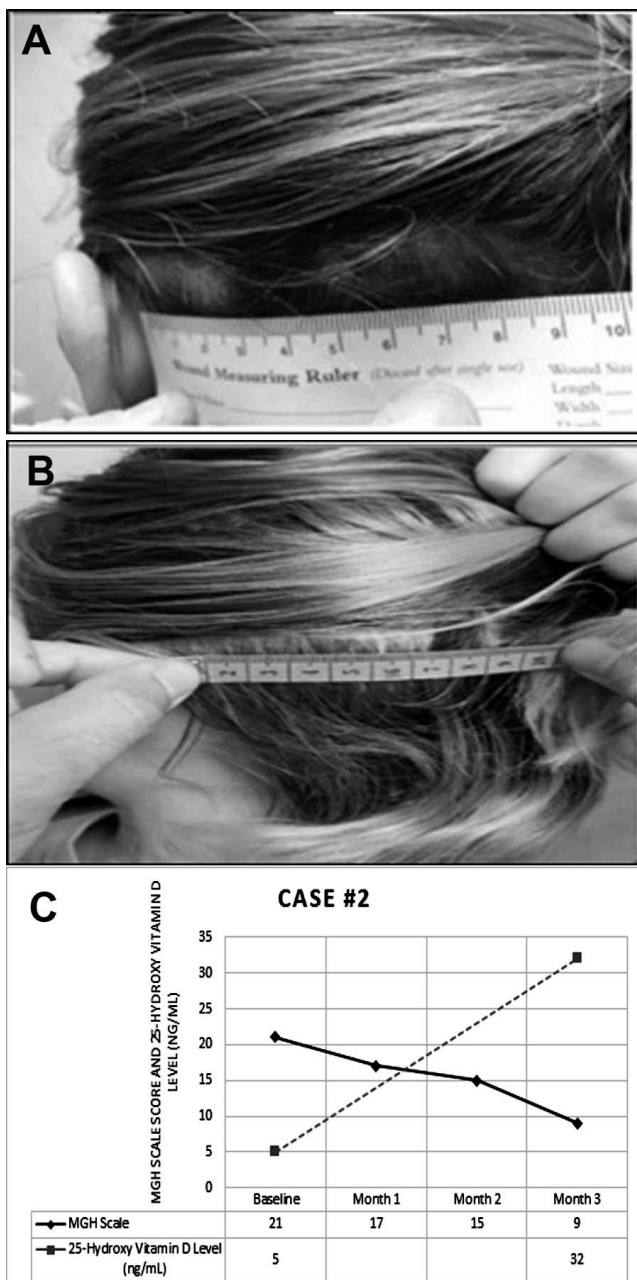


FIGURE 2: Patient Two before and after images and correlation analysis between hair pulling behavior and vitamin D supplementation. Hair density and distribution patterns for Patient Two are evident. (A) Shows the hair appearance in the first consultation, where we observed diffuse and scattered hair distribution indicating hair loss at the occipital region. (B) Shows the distribution and density of hair from the same occipital area for Patient Two during the last consultation. As shown, there is improvement in hair growth at the occipital region of the scalp after initiation of clomipramine 25 mg and vitamin D₃ 1000 IU daily. (C) Shows data from the Massachusetts General Hospital Scale for Hair Pulling and serum measurement for vitamin D pretreatment and posttreatment.

system.¹¹ Studies^{12,13} involving vitamin D suppression in mice have found behavioral changes involving anxiety-like symptoms. Studies^{11,14} in rats demonstrated associations between vitamin D and the production of the monoamine neurotransmitters serotonin, norepinephrine, and dopamine. Additional studies suggest a relationship between vitamin D, stress, and cortisol with in vitro evidence of a connection between vitamin D receptors and glucocorticoid receptors in the hippocampus.¹⁵ Vitamin D was also shown to be neuroprotective by inhibiting proinflammatory cytokines to modulate inflammation and proteins that reduced reactive oxygen species production.^{2,16}

Interestingly, in recent years a number of research and retrospective studies suggest a correlation between the serum levels of active vitamin D, the 25-hydroxyvitamin D form, and the prevalence of OCD. For example, in a study by Celik et al,¹⁷ vitamin D serum levels of 33 children with OCD manifesting as Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS) were compared with those of 20 healthy children. The authors reported that vitamin D deficiency was more common in the PANDAS group than in the healthy control group at a frequency of 48.5% for the PANDAS group compared to 20% for the control group, suggesting a possible role for vitamin D involvement in the pathophysiology.¹⁷ Further, Esnafoglu and Yaman¹⁸ found a similar trend of a deficiency in the serum level of vitamin D upon comparing 52 children and adolescent OCD patients with 30 controls. Their study included the use of the Yale-Brown Obsessive-Compulsive Scale to establish a correlation with vitamin D deficiency. Similarly, Yazici et al¹⁹ employed a semistructured study to analyze patients with OCD. Vitamin D serum levels in patients with OCD were compared to controls, and lower amounts of vitamin D in the patient group with OCD were observed, although a significant difference between the groups was not apparent. Taken together, these studies suggest initial evidence of a correlation between the serum levels of vitamin D and possible phenotypic development or manifestation of different types of OCD substantiating our approach.

The pathophysiology of TTM is not well understood. Neuroimaging has suggested disorganization of neurocircuitry in various brain regions, but neurotransmitter involvement has not been determined.^{2,16} Though the pathophysiologic cause of TTM may be poorly understood, it is well known that a psychological etiology exists for hair pulling that includes emotional causes such as stress and anxiety.²⁰ It is thought that hair pulling provides a means of escape from an unpleasant experience and the temporary relief from the associated negative feelings

may perpetuate hair pulling through a negative reinforcement cycle.²⁰

In addition to reducing the urge to pull, it is believed vitamin D supplementation significantly improved hair regrowth, which is often not seen after treatment with antidepressants or other medications alone. Studies have identified the use of vitamin D₃ analogs to stimulate hair regrowth.²¹ Evidence for this comes from animal models such as the mouse model of chemotherapy-induced alopecia, where topical application of the bioactive form of vitamin D resulted in hair regrowth.²² Further, experimental evidence using cellular models of alopecia and hair growth studies suggested complex molecular pathways downstream of vitamin D that regulate the ability of the dermal papillary cells to stimulate the surrounding epithelial stem cells for hair growth. For example, Aoi et al²³ reported the activation of the well-known tumor growth factor β /Wnt signaling pathway in human dermal papilla cells grown in culture following in vitro exposure to vitamin D. These studies provide anecdotal support for the use of vitamin D in the treatment of TTM.

Conclusion

Vitamin D supplementation for TTM is not documented in the medical literature. This case presents 2 patients who benefited from vitamin D supplementation with concomitant clomipramine resulting in a reduction in the urge to pull and in hair regrowth after only 3 to 4 months. We theorize that vitamin D supplementation may promote hair regrowth as well as target the underlying urge to pull through a mechanism that is not currently understood. Our approach to TTM therapy with vitamin D supplementation provides a potential new treatment option. Further research is necessary to confirm the proposed effects of vitamin D supplementation on targeting the urge to pull hair at a cellular level.

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