The role of vitamin D in women with hair loss in Babylon province

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Abstract:

Background: Hair loss is a common problem faced by many women,which has a variety of risk factors. Among them, Vitamin D3 has a key role to play in various disorders.Some recent evidence indicates an inconsistent association between Vitamin D3 deficiency and hair loss in women.

Aim of the study: The aim of this study was to compare the status of serumVitamin-D3 levels between the patients with hair fall and healthy people, as case & control group.

Method: The study was carried out on patients collected from Marjan Medical city in Babylon province. The practical side of the study was performed at the laboratory of biochemistry department at College of pharmacy/ University of Babylon. In total 15 patients with hair loss were finalized as the case group and another 15 healthy people were selected as the control group participants. All the case group patients were subjected to detailed history taking and examination to detect level of vitamin D3 , Blood samples were taken from both the group and to assess serum levels of Vitamin-D3. All data were processed, analyzed, and disseminated by Mini vidas.

Result: The mean ± SD level for Vitamin D3 level in patient and control group was (12.47±5.59) and (45.08±15.88), respectively showed a highly significant decrease between the two groups in terms of Vitamin D3 serum levels (P =0.0001).

Conclusions: Women with low serum vitamin D levels have a high potential for the development of FPHL suggesting that vitamin D may have a possible role in the etiopathogenesis of this pattern of hair loss.

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UV	Ultraviolet.
UVB	Ultraviolet B.
PTH	Parathyroid hormone.
AMP	Adenosine monophosphate.
VDR	Vitamine d receptors.
VDREs	Vitamine D receptors elements.
RXR	Retinoid x receptor.
MD	Mediterranean diet.
IU	International unite.
mcgs	Micrograms.
UK	United kingdom.
NHS	National health service.
VITD	VIDAS 25 OH Vitamin D TOTAL.
SPR	Solid phase receptacle.
ALP	Alkaline phosphate.
FPHL	Female Pattern Hair Loss.

1. Introduction:

1.1 Vitamin D:

Vitamin D (also referred to as calciferol) is a fat-soluble vitamin that is naturally present in a few foods, added to others, and available as a dietary supplement. It is also produced endogenously when ultraviolet (UV) rays from sunlight strike the skin and trigger vitamin D synthesis.

Vitamin D obtained from sun exposure, food (meet, milk ,cheese,…ext)s, and supplements is biologically inert and must undergo two hydroxylation in the body for activation .

Vitamin D that has long been known to help the body absorb and retain calcium and phosphorus; both are critical for building bone. Also, laboratory studies show that vitamin D can reduce cancer cell growth, help control infections and reduce inflammation. Serum concentration of 25(OH)D is currently the main indicator of vitamin D status. [1]

In serum, 25(OH)D has a fairly long circulating half-life of 15 days [1]. Serum concentrations of 25(OH)D are reported in both nanomoles per liter (nmol / L) and nanograms per milliliter (ng/mL).

Assessing vitamin D status by measuring serum 25(OH)D concentrations is complicated by the considerable variability of the available assays (the two most common ones involve antibodies or chromatography) used by laboratories that conduct the analyses[2,3].

As a result, a finding can be falsely low or falsely high, depending on the assay used and the laboratory. The international Vitamin D Standardization Program has developed procedures for standardizing the laboratory measurement of 25(OH)D to improve clinical and public health practice [2,4-5].

In contrast to 25(OH)D, circulating 1,25(OH)2D is generally not a good indicator of vitamin D status because it has a short half-life measured in hours, and serum levels are tightly regulated by parathyroid hormone, calcium, and phosphate [1]. Levels of 1,25(OH)2D do not typically decrease until vitamin D deficiency is severe [6].

1.1.2 Physiology:

Vitamin is synthesized in the skin epidermis through a series of chemical reactions that occur when 7-dehydrocholesterol, a type of cholesterol present in the skin, is exposed to sunlight UVB radiation. The UVB radiation transforms 7-dehydrocholesterol into cholecalciferol[7].

When cholecalciferol reach to the liver (25_ hydroxylase) which is an enzyme present in it , give a hydrogen atom to 25 the carbon of cholecalciferol molecule converting it to (25_ hydroxycholecalciferol) in another term (calcifediol)[7]. After hydroxylation in the liver (calcifediol) enter the kidney and undergo to another enzymatic reaction by (1_α hydroxylase) which give a hydrogen atom to the first carbon of calcifediol converting it to (1,25 hydroxycholecalciferol) in another term it's called (calcetriol) the active metabolite can enter the cell, bind to the vitamin D-receptor and subsequently to a responsive gene such as that of calcium binding protein[7].

After transcription and translation the protein is formed, e.g. osteocalcin or calcium binding protein. The calcium binding protein mediates calcium absorption from the gut.

The production of calcetriol is stimulated by parathyroid hormone (PTH) and decreased by calcium.

The active metabolite calcetriol has its effects through the vitamin D receptor leading to gene expression, e.g. the calcium binding protein or osteocalcin or through a plasma membrane receptor and second messengers such as cyclic AMP, The latter responses are very rapid and include the effects on the pancreas, vascular smooth muscle and monocytes[7].

The active metabolite has an anti-proliferative effect and downregulates inflammatory markers. Extra renal synthesis of calcetriol occurs under the influence of cytokines and is important for the paracrine regulation of cell differentiation and function. This may explain that vitamin D deficiency can play a role in the pathogenesis of auto-immune diseases such as multiple sclerosis and diabetes type 1, and cancer.

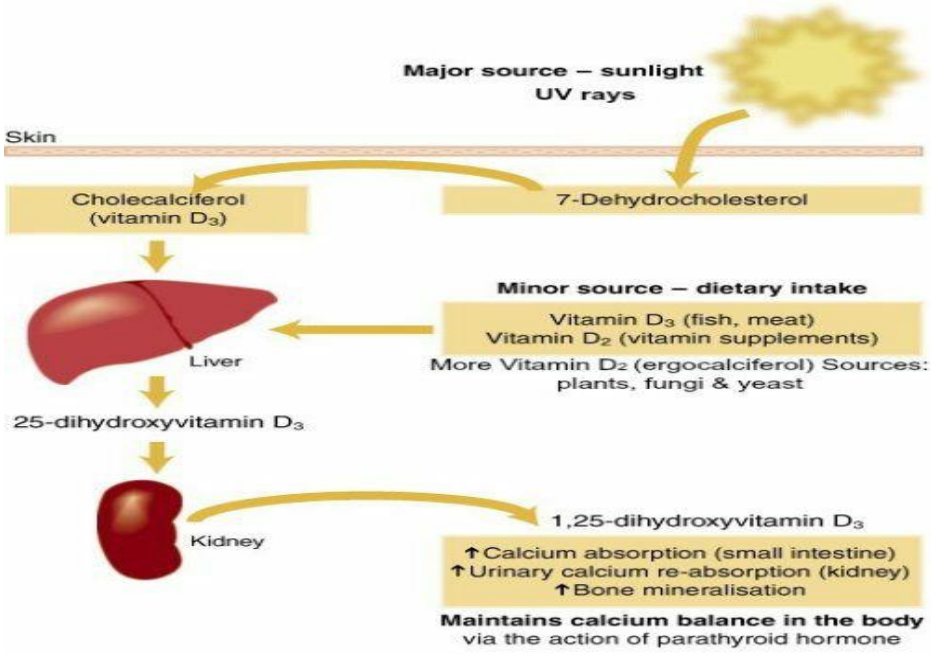

Figure 1.1 : Clinical physiology of vitamin D.

1.1.3 Type of vitamin D:

Vitamin D are available in several forms , as in the following table:

Table 1.1: Type of vitamin D.

Vitamin D_1	Ergocalciferol with lumisterol
Vitamin D_2	Ergocalciferol derived from ergosterol
Vitamin D_3	Cholecalciferol derived from 7- dehydrocholesterol in the skin
Vitamin D_4	22- dihydroergocalciferol
Vitamin D_5	Sitocalciferol derived from 7-dehydrositosterol

The two major forms are vitamin D2 and vitamin D3. Vitamin D2 (ergocalciferol) is largely human-made and added to foods, whereas vitamin D3 (cholecalciferol) is synthesized in the skin of humans from 7-dehydrocholesterol and is also consumed in the diet via the intake of animal-based foods. Both vitamin D3 and vitamin D2 are synthesized commercially and found in dietary supplements or fortified foods. The D2 and D3 forms differ only in their side chain structure. The differences do not affect metabolism (i.e., activation), and both forms function as prohormones[8].

1.1.4 Function:

1_Helps to retain calcium and phosphorus.

2_Reduces cancer cell growth.

3_Helps to control infections and reduces inflammation.

4_Maintains healthy bone and teeth.

5_Improves immunity.

6_Reduces the risk of autoimmune diseases.

7_Helps to strengthen muscles.

8_Provides strength and support to the skeletal frame of the body.

9_Reduces obesity and maintains proper weight.[9,10,11,12].

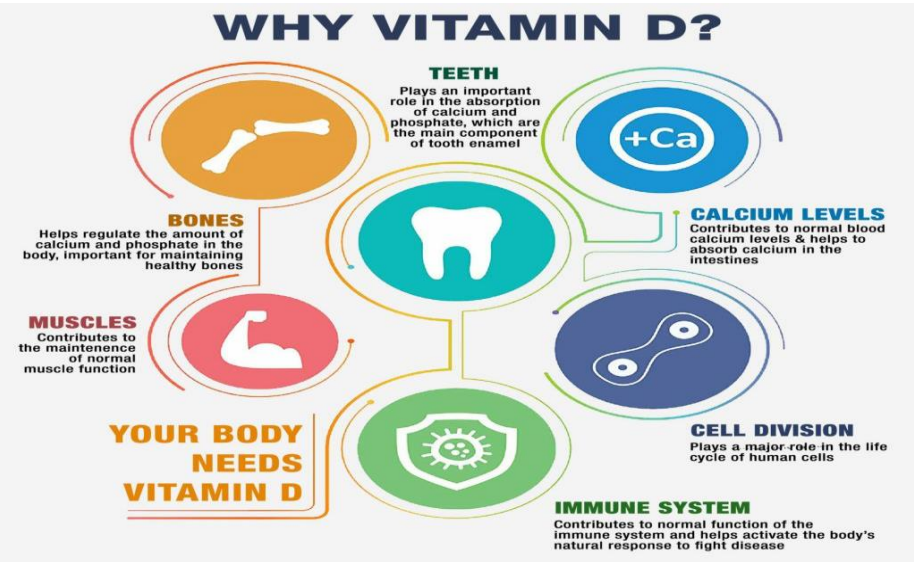

Figure 1.2 : Function of vitamin D

1.1.5 Mechanism:

Most, if not all, actions of vitamin D are mediated through a nuclear transcription factor known as the vitamin D receptor (VDR) (13). Upon entering the nucleus of a cell, 1α,25-dihydroxyvitamin D binds to the VDR and recruits another nuclear receptor known as retinoid X receptor (RXR). In the presence of 1α,25-dihydroxyvitamin D, the VDR/RXR complex binds small sequences of DNA known as vitamin D response elements (VDREs) and initiates a cascade of molecular interactions that modulate the transcription of specific genes. Thousands of VDREs have been identified throughout the genome, and VDR activation by 1α,25-dihydroxyvitamin D is thought to directly and/or indirectly regulate 100 to 1,250 genes [14].

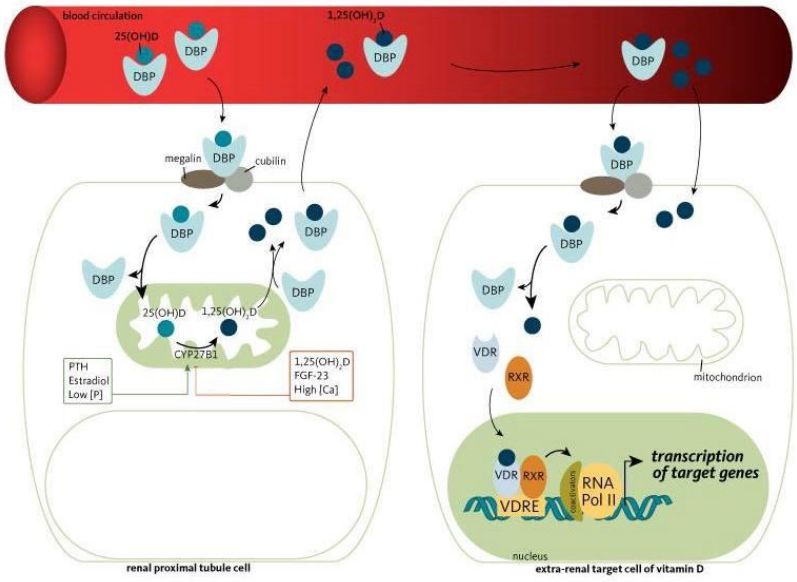

Figure 1.3: Mechanism of vitamin D.

1.1.6 Deficiency:

People can develop vitamin D deficiency when usual intakes are lower over time than recommended levels, exposure to sunlight is limited, the kidneys cannot convert 25(OH)D to its active form, or absorption of vitamin D from the digestive tract is inadequate. Diets low in vitamin D are more common in people who have milk allergy or lactose intolerance and those who consume an ova-vegetarian or vegan diet [1].

In children, vitamin D deficiency is manifested as rickets, a disease characterized by a failure of bone tissue to become properly mineralized, resulting in soft bones and skeletal deformities [15]. In addition to bone deformities and pain, severe rickets can cause failure to thrive, developmental delay, hypocalcemia seizures, tetanic spasms, cardiomyopathy, and dental abnormalities [16,17].

Prolonged exclusive breastfeeding without vitamin D supplementation can cause rickets in infants, and, in the United States, rickets is most common among breastfed Black infants and children [18].

Possible explanations for this increase include genetic differences in vitamin D metabolism, dietary preferences, and behaviors that lead to less sun exposure [16,17].

In adults and adolescents, vitamin D deficiency can lead to osteomalacia, in which existing bone is incompletely or defectively mineralized during the remodeling process, resulting in weak bones [17]. Signs and symptoms of osteomalacia are similar to those of rickets and include bone deformities and pain, hypocalcemia seizures, tetanic spasms, and dental abnormalities [16].

Screening for vitamin D status is becoming a more common part of the routine laboratory bloodwork ordered by primary-care physicians, irrespective of any indications for this practice [6,19-20].

1.1.7 Management of Vitamin D Deficiency:

The amount of vitamin D required to treat the deficiency depends largely on the degree of the deficiency and underlying risk factors.

- supplementation for 8 weeks with Vitamin D3, either 6,000 IU daily or 50,000 IU weekly, can be considered.[13] Once the serum 25-hydroxyvitamin D level exceeds 30 ng/mL, a daily maintenance dose of 1,000 to 2,000 IU is recommended.
- A higher-dose initial supplementation with vitamin D3 at 10,000 IU daily may be needed in high-risk adults who are vitamin D deficient (African Americans, Hispanics, obese, taking certain medications, malabsorption syndrome). Once serum 25-hydroxyvitamin D level exceeds 30ng/mL, 3000 to 6000 IU/day maintenance dose is recommended.
- Children who are vitamin D deficient require 2000 IU/day of vitamin D3 or 50,000 IU of vitamin D3 once weekly for 6 weeks. Once the serum 25(OH)D level exceeds 30 ng/mL, 1000 IU/day maintenance treatment is recommended. According to the American Academy of Pediatrics, breastfed infants and children who consume less than 1 L of vitamin D-fortified milk need 400 IU of vitamin D supplementation.
- Calcitriol can be considered where the deficiency persists despite treatment with vitamin D2 and/or D3. The serum calcium level shall be closely monitored in these individuals due to an increased risk of hypercalcemia secondary to calcitriol.

- Calcidiol can be considered in patients with fat malabsorption or severe liver disease.

1.2 Hair loss:

Hair loss or alopecia, a clinical condition that is frequently seen in dermatology clinics, can be caused by many etiological factors and it significantly affects the patients' quality of life [21]. This group of diseases is basically divided in two subgroups: cicatricial alopecia's and non-cicatricial alopecia's.

While cicatricial alopecia may progress with loss of follicles, thus causing irreversible hair loss, this condition is usually reversible in non-cicatricial ones. Many causes are known to have a role in non-cicatricial alopecia, including emotional issues, chronic disorders, dietary inadequacies, trace elements, and vitamin deficiencies [22]. Other factors can be stress, drug use, immune system, endocrine disorders, and genetic and epigenetic changes [23].

A balanced and regular diet is very important for healthy hair: sudden weight loss, low-caloric diets, unbalanced diet, obesity, and excessive intake of vitamin and mineral supplements can cause hair loss. Micronutrients, which are the main elements of the hair follicle cycle, are very important in alopecia, which is why dietary supplements (mostly vitamin and mineral) are among the preferred methods to prevent hair loss. Given the frequency of hair loss in current times and its impact on the patients' social lives, finding effective alopecia treatments impacts a huge portion of the population [22].

The role of diet in the development and treatment of alopecia has recently been a hot topic of research. It has been found that plant-rich diets – such as the Mediterranean Diet (MD), whose main nutrients are rich in antioxidants, anti-inflammatory, and

estrogenic components – include chemicals that stimulate hair growth and reduce hair loss. These diets contain phytochemicals that promote hair development by lowering the generation of reactive oxygen species in the dermal papilla cells, causing growth hormones to be secreted [24].

What the role of vitamin D Plays in Hair Growth?

Believe it or not, vitamin d isn't actually a nutrient in the same way as vitamin a, b12, or c. Actually, it's a hormone. In your body, vitamin d is metabolized in skin cells called keratinocytes. Keratinocytes are absolutely vital in skin repair, muscle growth, and the health of hair follicles .

Your hair is made of a protein called Keratin which is made by Keratinocytes. Keratinocyte cells are also found right at the top of your dermic layer, right where your hair follicles are. So, with enough vitamin d in the Diet and lifestyle, your hair should always be happy and healthy.

When your body doesn't have enough vitamin d, your keratinocytes seem to struggle regulating your natural hair cycle, reducing hair growth and increasing hair shedding.

With lower vitamin d levels, the anagen (growth) phase of your hair cycle seems to shorten and your telogen (resting) phase seems to lengthen. Not to mention, your dermic layer (the foundations that hold your hair in the skin) is weakened, making your hair more likely to fall out.

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Most governments and healthcare systems across the world will recommend a daily dose of at least 600 IU (international units) or 15 mcgs (micrograms). Furthermore, the older you get, the more vitamin d you should expose yourself to; at least 800 IU is recommended for people over 70.

As well as vitamin d supplements, you can also intake this helpful hormone through a number of different methods. As well as sunlight exposure in the warmer months, dairy and protein are foods naturally high in vitamin d.

If you want to promote healthy hair growth without a vitamin d supplement, the NHS recommends introducing the following foods into your diet:

- oily or fatty fish – such as salmon, sardines, herring and mackerel.
- red meat.
- liver.
- egg yolks.
- fortified foods – such as some fat spreads and breakfast cereals.

1.2.1 Factor or Reason:

Vitamin D deficiency can result from several causes.

1. Decreased dietary intake and/or absorption: Certain malabsorption syndromes such as celiac disease, short bowel syndrome, gastric bypass, inflammatory bowel disease, chronic pancreatic insufficiency, and cystic fibrosis may lead to vitamin D deficiency. Lower vitamin D intake orally is more prevalent in the elderly population. [25]

2. Decreased sun exposure: Twenty minutes of sunshine daily with over 40% of skin exposed is required to prevent vitamin D deficiency.[26] Cutaneous synthesis of vitamin D declines with aging. Dark-skinned people have less cutaneous vitamin D synthesis. Decreased exposure to the sun, as seen in individuals who are institutionalized or have prolonged hospitalizations, can also lead to vitamin D deficiency.[27] Effective sun exposure is decreased in individuals who use sunscreens consistently.

3. Decreased endogenous synthesis: Individuals with chronic liver disease such as cirrhosis can have defective 25-hydroxylation, leading to deficiency of active vitamin D. Defects in 1-alpha 25-hydroxylation can be seen in hyperparathyroidism, renal failure, and 1-alpha hydroxylase deficiency.

4. Increased hepatic catabolism: Medications such as phenobarbital, carbamazepine, dexamethasone, nifedipine , spironolactone, clotrimazole , and rifampin induce hepatic p450 enzymes, which activate the degradation of vitamin D.[28]

5. End organ resistance: End-organ resistance to vitamin D can be seen in hereditary vitamin D-resistant rickets.

1.2.2 Complication:

The most serious complications of vitamin D deficiency include:

_ Low blood calcium levels (hypocalcemia).

_ Low blood phosphate levels (hypophosphatemia).

_ Rickets (softening of bones during childhood).

_ Osteomalacia (softening of bones in adults).

All of these conditions are treatable. While rickets is a treatable and often curable disease, treating it as soon as possible is important. When not treated, milder cases of rickets can result in long-term bone damage that can keep bones from growing properly.

Severe cases that aren't treated can lead to seizures, heart damage and death. However, these complications have become less common over time because many foods and drinks have added vitamin D.

vitamin D insufficiency is associated with reduced bone density (osteopenia or osteoporosis), a mild decrease of the blood calcium level, elevated parathyroid hormone (which accelerates bone resorption), an increased risk of falls, and possibly fractures, all of which can seriously affect a person's quality of life.

Thus, identifying and treating vitamin D insufficiency or deficiency is important to maintain bone strength. Treatment may even improve the health of other body systems, such as the immune, muscular, and cardiovascular systems.

1.2.3 Materials:

Subjects and samples:

Subjects group:

The study was carried out on patients collected from Marjan Medical city in Babylon province. The practical side of the study was performed at the laboratory of biochemistry department at College of pharmacy/ University of Babylon.

This study was included 30 female the age of them between (18-65) years old, which were divided into two groups:

♦ Group A (control group) : which included 15 women with no hair loss.

♦ Group B (patient): which included 15 women with hair loss.

1.2.4.Instruments and tools:

All instruments and tools that have been used in this study are listed in the table below:

***The device used in this study is VIDAS.**

Table (2.1): Instruments and tools used in this study:

No	Instruments	Company and country
1	Centrifuge	Sigma (Germany)
2	Disposable syringe	EASYMED/ China
3	Disposable yellow tips	China
4	Disposable blue tips	China
5	Disposable test tube	Afco-despo/ Jordan
6	Eppindorf tube	Slamed /Germany
7	Freezer	Liebhe (Austria)
8	Micropipettes 100-1000 µL	XINKANG / China
9	Micropipettes 5-50 µl	XINKANG / China
10	Spectrophotometer	England

1.2.5.Principle :

The assay principle combines an enzyme immunoassay competition method with a final fluorescent detection (ELFA). ®

The Solid Phase Receptacle (SPR) serves as the solid phase as well as the pipetting device for the assay. Reagents for the assay are ready-to-use and pre- dispensed in the sealed reagent strips.

All of the assay steps are performed automatically by the instrument. The reaction medium is cycled in and out of the SPR several times.

The sample is mixed with pre-treatment reagent to separate vitamin D from its binding protein

The pre-treated sample is then collected and transferred into the well that contains an alkaline phosphatase (ALP)- labeled anti-vitamin D antibody (conjugate).

The vitamin D antigen present in the sample and the vitamin D antigen coating the interior of the SPR compete for binding sites on the anti-vitamin D antibody-ALP conjugate.

During the final detection step, the substrate (4-Methyl- umbelliferyl phosphate) is cycled in and out of the SPR. The conjugate enzyme catalyzes the hydrolysis of this substrate into a fluorescent product (4-Methyl- umbelliferone), the fluorescence of which is measured at 450 nm. The intensity of the fluorescence is inversely proportional to the concentration of vitamin D antigen present in the sample. At the end of the assay, results are automatically calculated by the instrument in relation to the calibration curve stored in memory, and then printed out. [29]

1.2.6.Procedure:

1.Only remove the required reagents from the refrigerator. They can be used immediately.

2. Use one "VITD" strip and one "VITD" SPR® from the kit for each sample, control or calibrator to be tested. Make sure the storage pouch has been carefully resealed after the required SPRs have been removed.

3. The test is identified by the "VITD" code on the instrument. The calibrator must be identified by "S1", and tested in duplicate. If the control is to be tested, it should be identified by "C1".

4. If necessary, clarify the samples by centrifugation.

5. Mix the calibrator, control and samples using a vortex- type mixer (for serum or plasma separated from the pellet).

6. Before pipetting ensure that samples, calibrators, controls and diluent are free of bubbles.

7. For this test, the calibrator, control, and sample test portion is 100 µL.

8. Insert the "VITD" SPRs and "VITD" strips into the instrument. Check to make sure the color labels with the assay code on the SPRs and the Reagent Strips match.

9. Initiate the assay as directed in the User's Manual. All the assay steps are performed automatically by the instrument.

10.Reclose the vials and return them to 2–8°C after pipetting.

11.The assay will be completed within approximately 40 minutes. After the assay is completed, remove the SPRs and strips from the instrument.

12.Dispose of the used SPRs and strips into an appropriate recipient.[30]

Table 2.2. Mean± SD of levels of Vit.D3 in normal patient (no hair loss), Vit.D3 in patient (with hair loss).

Biochemical test	Mean± SD (control group)	Mean± SD (patient)	P value
Vit.D3	45.08±15.88	12.47±5.59	0.0001

P ≤ 0.01 Significant .

Normal level of Vitamin D3 (30-100 ng/ml).

Discussion:

Vitamin D is a factor that has recently been considered in dealing with these patients. The purpose of this study was to evaluate the serum levels of Vitamin D in patients with FPHL and compare it with healthy controls.

In this case-control study, 15 women with Femal Pattern Hair Loss (FPHL) were evaluated as well as the same number of healthy women matched for age, hours spent under sunlight per day, and body mass index. Serum 25(OH) D3 level was measured using VIDAS. [30,31] The presentstudy matched with Hoda Moneib et al.30 and H.Rasheed et al.31 Accordingly, a screening test forVitamin D level would be a useful measure in women with hair loss, and dietary supplementsof Vitamin D may help to treat these patients.

In This study the Mean (SD) serum Vitamin D3 level in patient and control group was (12.47±5.59) and (45.08±15.88), respectively showed a highly significant decrease between the two groups in terms of Vitamin D3 serum levels (P =0.0001). The results of the study support the suggestion that the decrease in hair density is associated with altered levels of serum vitamin D. deficiency rather than insufficiency was more common among females suffer from hair loss. The severity of hair loss was directly correlated with the degree of vitamin D deficiency. Some study show the Longer the duration of hair loss the more sever vitamin D deficiency. [32,33]

This finding should encourage to measure vitamin D level at an early stage of hair loss with the hope of reducing or preventing the progression of alopecia, also dietary supplements of vitamin D may help to treat these patients.

It is worth mentioning that most Iraqi women are at risk of developing vitamin D deficiency due to lack of sufficient sunlight exposure and the wearing of modest clothing that covers most of the body, in addition to the lack of eating for rich in vitamin D.

Conclusions:

This study indicated the correlation between the incidence of FPHL and decreased serum levels of Vitamin D3. It is recommended to evaluate serum Vitamin D3 levels as well as other hormone assays in these patients.[34]

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الخلاصة:

الخلاصة: يعد تساقط الشعر مشكلة شائعة تواجهها العديد من النساء، ولها مجموعة متنوعة من عوامل الخطر. من بينها، يلعب فيتامين د دورًا رئيسيًا في العديد من الاضطرابات. تشير بعض الأدلة الحديثة إلى وجود علاقة غير متناسقة بين نقص فيتامين د3 وفقدان الشعر لدى النساء.

هدف الدراسة: كان الهدف من هذه الدراسة هو مقارنة حالة مصل مستويات فيتامين د بين المرضى الذين يعانون من تساقط الشعر والأشخاص الأصحاء، كمجموعة الحالات ومجموعة السيطرة.

الطريقة: أجريت الدراسة على المرضى الذين تم جمعهم من مدينة مرجان الطبية في محافظة بابل . تم تنفيذ الجانب العملي للدراسة في مختبرقسم الكيمياء الحيوية في كلية الصيدلة/ جامعة بابل. في المجموع 15 تم الانتهاء من المرضى الذين يعانون من تساقط الشعر كمجموعة حالة و15 شخصًا أصحاء آخرين تم اختيارهم كمشاركين في المجموعة الضابطة. و جميع مرضى مجموعة الحالة خضعوا لأخذ التاريخ المرضي التفصيلي والفحص للكشف عن مستوى فيتامين د3،تم م أخذ عينات الدم من كلا المجموعتين لتقييم مستويات فيتامين د3 .تمت معالجة البيانات وتحليلها ونشرها بواسطة mini vidas.

النتيجة: mean ± SD لمستوى فيتامين د في المريض والمجموعة المسيطرة (12.47±5.59) و(45.08±15). على التوالي أظهرت انخفاضا كبيرا بين المجموعتين من حيث مستويات مصل فيتامين د P=0.0001.

الاستنتاج : النساء اللاتي لديهن مستويات منخفضة من فيتامين د في الدم لديهن احتمالية عالية في تطور FPHL مما يشير الى ان فيتامين د له دور محتمل في التسبب في هذا النمط من تساقط الشعر.

دور فيتامين د لدى النساء المصابات بتساقط الشعر في محافظة بابل

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