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Impact of Inadequate Vitamind D Status on Anemia in Pregnant Females

Marwa, A. Bumaluma[1 *], Aziza, M. Alsheekh, [2], Haleemah Abdullah Abu-BakerJibreel[3]

[1, 2, 3,] College of Medical Technology, Lab Medicine, Derna, Libya

[*]Corresponding author: marwabomaloma16@gmail.com

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Abstract:
Vitamin D and iron deficiencies are two major global health concerns, particularly among pregnant women, where they may contribute to adverse maternal and fetal outcomes. This study aimed to investigate the relationship between vitamin D deficiency and anemia in pregnant women in Derna, Libya. A cross-sectional design was employed, involving 126 pregnant women aged 18–45 years. Serum levels of vitamin D, hemoglobin, and ferritin were measured and analyzed using SPSS. The findings revealed a high prevalence of vitamin D deficiency, with 69% of participants exhibiting severe deficiency (<20 ng/mL). Anemia was also widespread, affecting 91.3% of the sample, while 38.1% had low ferritin levels. Statistically significant positive correlations were observed between vitamin D levels and both hemoglobin ($r = 0.269$, $p = 0.002$) and ferritin ($r = 0.465$, $p = 0.001$), suggesting that vitamin D deficiency may contribute to anemia by influencing iron homeostasis, possibly through hepcidin regulation and anti-inflammatory mechanisms. The results emphasize the importance of assessing vitamin D status during pregnancy as part of routine care. Future longitudinal and interventional studies are recommended to confirm the causal relationship and evaluate the impact of vitamin D supplementation on anemia management in pregnant populations.

Keywords: Vitamin D deficiency, anemia, Pregnancy, Ferritin, Hemoglobin, Hepcidin, Libya

Introduction

Vitamin D deficiency or insufficiency affects over one billion people globally. The need for vitamin D supplementation should be assessed based on various factors such as lifestyle, climate, sun exposure, and diet. Iron deficiency anemia (IDA) impacts more than two billion individuals worldwide. In India, the prevalence of IDA was reported at 53% among women aged 15-49 years in 2015-2016, primarily due to iron loss from menstruation. This requirement increases during pregnancy due to the heightened demand for nutrients and the larger blood volume necessary for fetal development. (Basutkar RS et al 2019). Iron is crucial for maintaining the body's homeostasis and energy production. The global effort to manage IDA has focused significantly on iron supplementation. Recent research indicates a link between iron and vitamin D pathways. Vitamin D deficiency may lead to increased levels of Hepcidin, a molecule that reduces iron absorption and traps iron within tissues. Moreover, vitamin D receptors are found in nearly all cells, including bone marrow progenitor cells. Recent findings have shown that calcitriol can enhance the expression of erythropoietin receptors, thereby stimulating erythropoiesis (Basutkar RS et al 2019) The World Health Organization (WHO) defines anemia as having serum hemoglobin (Hgb) levels of less than 13 g/dL for men over 15 years, less than 12 g/dL for non-pregnant women over 15, and less than 11 g/dL for pregnant women. According to these criteria, over 30% of the global population is affected by anemia, with more than half of these cases attributed to iron deficiency. (Nur-Eke R, and Özen M 2020) Serum 25(OH)D is the most reliable indicator of vitamin D levels in the body. Although there is no consensus on the optimal level of serum 25(OH)D, it is generally considered sufficient when levels exceed 30 ng/mL (75 nmol/L), insufficient when between 20-30 ng/mL (50-75 nmol/L), and deficient when below 20 ng/mL (50 nmol/L). Additionally, serum 25(OH)D levels above 150 ng/mL may result in vitamin D intoxication. While vitamin D and iron are recognized as crucial for maternal health and fetal development during pregnancy, there is

limited understanding of their interrelationship and regulation during this period. To enhance existing knowledge, a systematic review and meta-analysis of all available observational studies assessing the link between vitamin D deficiency and anemia in pregnant women was conducted. The goal was to clarify the mechanisms by which vitamin D and iron are regulated during pregnancy and to evaluate their effects on both pregnant women and the fetus. Additionally, given that vitamin D deficiency and anemia are known risk factors for adverse pregnancy outcomes, this study also aims to explore the association between vitamin D deficiency and gestational anemia through observational research. (Lima MS, et al 2022) Vitamin D is a nutrient you need for good health. It helps your body absorb calcium, one of the main building blocks for strong bones. Together with calcium, vitamin D helps protect you from developing osteoporosis, a disease that thins and weakens the bones and makes them more likely to break. Your body needs vitamin D for other functions too. Your muscles need it to move, and your nerves need it to carry messages between your brain and your body. Your immune system needs vitamin D to fight off invading bacteria and viruses (National Institutes of Health. Office of Dietary Supplements 2023 Apr 19)

Synthesis and action of vitamin D 7-dehydrocholesterol (7-DHC), a precursor of vitamin D, is part of the Kandutsch-Russell cholesterol pathway. The conversion of 7-DHC to cholesterol is facilitated by the enzyme 7-dehydrocholesterol reductase, which is regulated by factors such as vitamin D and cholesterol. These factors promote the degradation of 7-DHC, leading to increased levels available for vitamin D synthesis. (Bikle DD 2015). Hepatic Production of 25-Hydroxyvitamin D (25OHD) . The hydroxylation of vitamin D2 and D3 to form 25OHD primarily occurs in the liver, although several other tissues can also perform this enzymatic function. 25OHD serves as the main circulating form of vitamin D and is a valuable clinical marker for assessing vitamin D status. (Bikle DD 2015). Action of Vitamin D he active form of vitamin D, 1,25(OH)2D, acts as a ligand for the vitamin D receptor (VDR), a transcription factor. The effects of 1,25(OH)2D are primarily mediated through vitamin D response elements (VDREs), which influence the expression of genes that contain specific DNA sequences known as VDREs. These elements can be located far from the gene's coding region. Some effects of 1,25(OH)2D occur more rapidly and may involve membrane-bound vitamin D receptors or actions outside the nucleus. Additionally, certain effects do not require the presence of 1,25(OH)2D

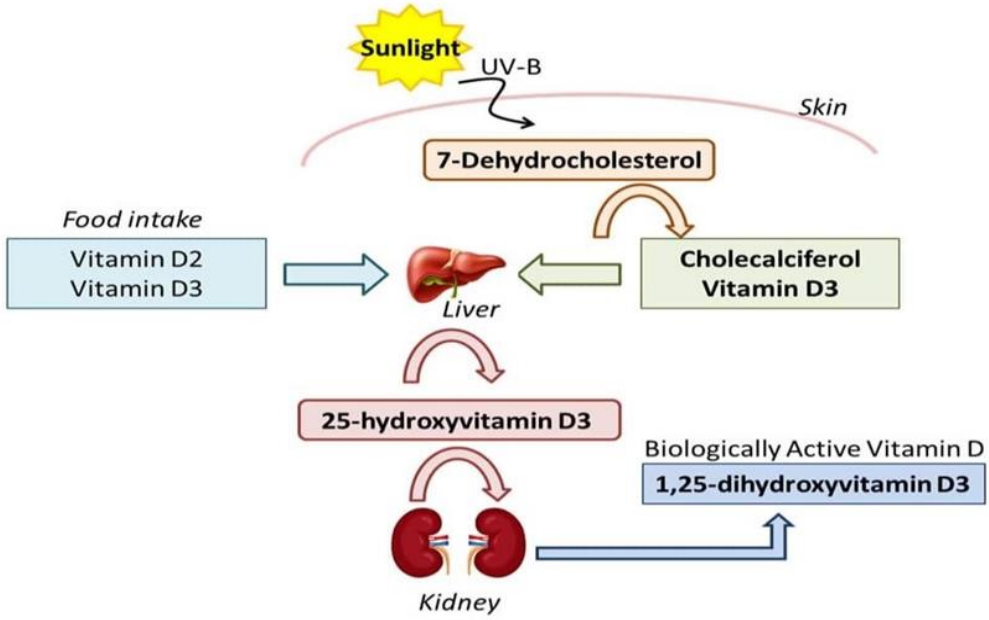


Figure:1 Pathway of Vitamin D Production (Aiello G, et al 2024)

Physiological functions of vitamin D

The primary physiological role of vitamin D is to maintain serum mineral balance, ensure bone quality, and support the overall health of extrarenal organs. The active form, $1,25(OH)_2D$, facilitates the absorption of calcium and phosphate through the transient receptor potential vanilloid type 5 and type 6 channels located in intestinal epithelial cells Serum parathyroid hormone (PTH) can stimulate the production of 1αhydroxylase in renal tissues, leading to increased synthesis of $1,25(OH)_2D$. However, elevated levels of $1,25(OH)_2D$ inhibit PTH secretion through feedback mechanisms. Additionally, hypocalcemia raises serum PTH levels, which enhances calcium

resorption and phosphate excretion, thereby promoting further synthesis of 1,25(OH)$_2$D in renal tubule cells. Consequently, serum levels of PTH, calcium, and phosphate collectively influence the synthesis of 1,25(OH)$_2$D to maintain calcium and phosphate homeostasis.]M(Liu WC et al 2016)oreover, 25(OH)D can be converted to active 1,25(OH)$_2$D in extrarenal cells, such as those in the brain, heart, and pancreas, without requiring kidneyinvolvement.Approximately 85% of circulating 25(OH)D is utilized in the local production of 1,25(OH)$_2$D through autocrine and paracrine mechanisms, with the concentration of extrarenal 1αhydroxylation potentially being higher in chronic kidney disease (CKD) than in normal conditions. (Liu WC et al 2016) Furthermore, the vitamin D receptor (VDR) is present in various organs, including immune, muscular, and nervous systems, as well as in solid organs like bones and kidneys. Thus, the extraskeletal functions of vitamin D encompass renal function preservation, cardiovascular protection, immune regulation, and cancer prevention, among others. (Liu WC et al 2016), Vitamin D levels and deficiency Serum 25-hydroxyvitamin D concentrations of 50 nmol/L or higher are regarded as optimal for vitamin D status. The prevalence of vitamin D deficiency varies based on definitions, with most literature agreeing on levels below 50 nmol/L. A recent article from the NutriProfiel project defines vitamin D deficiency as follows: for children aged 0-4 years, levels below 20 nmol/L; for those aged 5-64 years, levels below 30 nmol/L; and for individuals over 65 years, levels below 50 nmol/L. (Edis Z, and Haj Bloukh S 2016) as illustrated in Figure :2

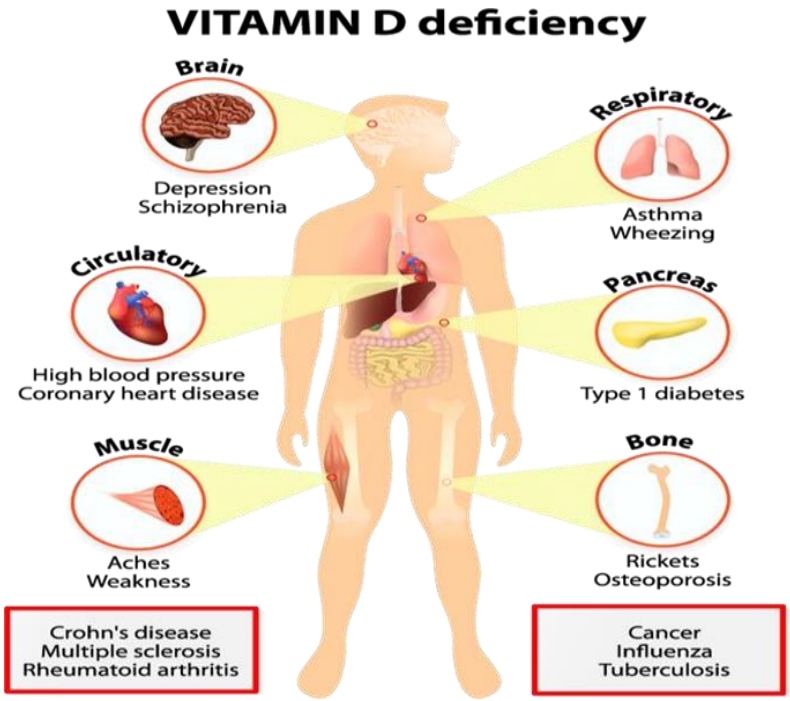


Figure 2: Vitamin D deficiency (Dastidar R, and Halder2017)

Vitamin D during pregnancy and lactation Vitamin D functions as a preprohormone with effects that go beyond calcium metabolism, particularly evident during pregnancy when its metabolism is less constrained. At no other point in life is there such a direct link between 25(OH)D3 and 1,25(OH)2D3 production, with the latter increasing over 2.5 times compared to nonpregnant levels. This rise in 1,25(OH)2D3 likely plays a role in immune regulation, although studies examining the impact of vitamin D status on immune function during pregnancy are limited. Additionally, there have been few randomized controlled trials to determine optimal vitamin D levels in pregnant women. (Christakos S et al 2013). Vitamin D and anemia Chronic inflammation and reduced glomerular filtration rate (GFR) in patients with chronic kidney disease (CKD) lead to iron imbalance and increased production of hepcidin. Vitamin D deficiency disrupts both innate and adaptive immune functions, resulting in elevated inflammatory cytokines such as IL-6, IFN-γ, and TNF-α. These cytokines activate the canonical Janus kinase (JAK)/signal transducer and activator of transcription (STAT) pathway, leading to the phosphorylation of JAKs and STAT3, which stimulates hepcidin expression in the liver . (Liu WC et al 2016). Hepcidin plays a crucial role in regulating iron homeostasis by limiting intestinal iron absorption and restricting the release of iron from

macrophages and liver stores through the degradation of ferroportin. In states of iron deficiency, anemia, or hypoxia, hepcidin production is inhibited, enhancing iron availability in the liver. Conversely, inflammation and excessive iron supplementation stimulate hepcidin production, which inhibits ferroportin activity and reduces iron availability. Several factors contribute to iron deficiency in CKD patients, including the use of phosphate binders and antacids, blood loss during hemodialysis, and the administration of erythropoiesis-stimulating agents (ESA). Additionally, secondary hyperparathyroidism directly inhibits erythroid progenitors, endogenous erythropoietin (EPO) synthesis, and red blood cell (RBC) survival. It also indirectly leads to bone marrow fibrosis, hyperphosphatemia, and increased serum alkaline phosphatase levels, all of which contribute to ESA hyporesponsiveness. (Liu WC et al 2016). Recent studies indicate that vitamin D deficiency, low hemoglobin levels, and ESA resistance act as pathophysiological contributors to renal anemia. Supplementing with vitamin D or its active forms has been associated with improvements in anemia and a reduction in ESA requirements. Consequently, there is an inverse relationship between vitamin D levels and ESA needs in CKD patients. Furthermore, vitamin D supplementation promotes anti-inflammatory effects and enhances erythroid proliferation. (Liu WC et al 2016). The association between vitamin D deficiency and anemia especially anemia of inflammation has been established in various observational and epidemiological studies. Several mechanisms may explain this association. One possibility is that vitamin D directly and indirectly suppresses hepcidin expression. Specifically, $1,25(OH)_2D_3$ interacts with vitamin D response elements on the promoter of the hepcidin gene in monocytes and hepatocytes, leading to reduced hepcidin mRNA transcription. Additionally, vitamin D may indirectly affect hepcidin levels by suppressing pro-inflammatory cytokines that stimulate hepcidin production during inflammation. Furthermore, $1,25(OH)_2D_3$ has been shown to support erythropoiesis by promoting the proliferation of burst-forming unit erythroid cells and enhancing erythroid progenitor cell proliferation synergistically with erythropoietin. In (Moran-Lev H, et al 2018) a study, serum 25-OHD levels were significantly lower, and hepcidin levels were significantly higher in children with infectious diseases and concurrent anemia compared to those without anemia or infection. A serum 25-OHD level below 20 ng/mL was associated with a significantly increased risk of anemia compared to levels above 20 ng/mL. However, no significant correlation was found between serum 25-OHD and circulating hepcidin levels. This may be due to the fact that while intracellular production of $1,25(OH)_2D_3$ in monocytes and macrophages is sensitive to serum 25-OHD availability, the hepcidin-ferroportin axis is regulated by various cytokines and Toll-like receptors. (Moran-Lev H, et al 2018) Moreover, hepcidin synthesis and release from hepatocytes and macrophages into circulation are controlled by at least eight different proteins. Interestingly, research by Adams et al. demonstrated that while $1,25(OH)_2D_3$ directly stimulates the expression of cathelicidin, another antibacterial protein, there was no correlation between serum 25-OHD levels and circulating cathelicidin levels. This highlights the significance of local intracrine mechanisms in regulating $1,25(OH)_2D_3$ and cathelicidin production. (Moran-Lev H, et al 2018) in

the relationship between vitamin D and anemia and to further clarify the in conclusion, the findings suggest that the hepcidin-ferroportin axis plays a role in the development of hypoferremia and iron-restrictive anemia in children with acute infectious diseases, indicating that low vitamin D status may contribute to anemia. (Moran-Lev H, et al 2018)

Material and Methods

Research Design

A cross-sectional study design will be utilized to evaluate vitamin D levels and hemoglobin, ferritin concentrations among pregnant participants across multiple healthcare facilities and laboratories. This approach enables the simultaneous measurement of both variables.

Sample

The target population includes pregnant women aged 18–45 attending antenatal clinics. A sample of 126 participants will be selected through convenience sampling. Inclusion criteria will consist of pregnant women who provide informed consent to participate.

Data Collection Tools

Data will be gathered using the following methods:

-Serum Vitamin D Levels: Measured using a standardized immunoassay. -Anemia Assessment: Hemoglobin levels will be determined via a complete blood count (CBC).

-Ferritin Level Analysis: Conducted using techniques such as immunoassay or ELISA (enzyme-linked immunosorbent assay).

Data Collection Procedures

Participants will be recruited during their routine antenatal visits, where blood samples will be collected to measure both serum vitamin D and hemoglobin levels.

Data Analysis

Data will be analyzed using the Statistical Package for Social Sciences (SPSS) version 26. Continuous variables will be presented as means and standard deviations, while categorical variables will be reported as frequencies

and percentages. Bivariate analysis will be employed to examine the relationships between ferritin levels, hemoglobin, and vitamin D status. A p-value of <0.05 will be considered statistically significant.

Ethical Considerations

Ethical approval will be secured from the Institutional Review Board (IRB). Informed consent will be obtained from all participants, ensuring they understand the study's purpose and their right to withdraw at any time without impacting their medical care

Results and discussion

A total of 126 pregnant women were included in this study. Among the pregnant women, 45.2% were aged 26–35 years, making this the most represented age group. This was followed by 28.6% aged ≤25 years, and 26.2% aged ≥36 years (Figure 3).

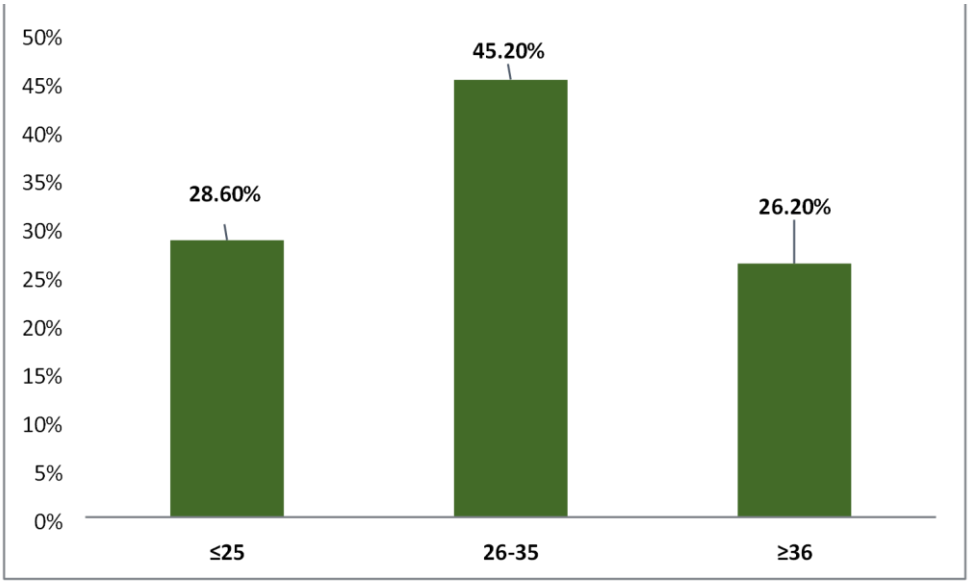


Figure 5: Age of pregnant women

Table 1: Average value, standard deviation, and range of age, Vitamin D, Ferritin and Hb

	Minimum	Maximum	Mean	Std.Deviation
Age	18	47	30.75	8.043
Vitamin D	3.0	60.0	16.59	13.449
Ferritin	3.0	134.5	24.09	19.564
Hb	7.0	12.2	9.84	1.1805

presents the descriptive statistics for maternal age, Vitamin D, ferritin, and hemoglobin (Hb) levels among the of pregnant women. The mean maternal age was 30.75 years (SD = 8.04), with a range of 18 to 47 years.
The average serum Vitamin D level was 16.59 ng/mL (SD = 13.45). Ferritin levels ranged from 3.0 to 134.5 ng/mL, with a mean of 24.09 ng/mL (SD = 19.56). The mean hemoglobin level was 9.84 g/dL (SD = 1.18), with values ranging from 7.0 to 12.2 g/dL (Table 1).

Table 2: Prevalence and Severity of Vitamin D Deficiency in Pregnant Women

	NO	Percentage %
Sever deficiency (<20 ng/ml)	87	69.0
Deficiency (20-30 ng/ml)	21	16.7
Insufficient (30-50 ng/ml)	12	9.5
Optimal (>51 ng/ml)	6	4.8

A majority, 69.0%, had severe deficiency with serum levels below 20 ng/mL, while 16.7% had levels between 20–30 ng/mL, indicating moderate deficiency. Additionally, 9.5% were classified as insufficient (30–50 ng/mL), and only 4.8% of participants had optimal vitamin D levels above 51 ng/mL (Table 2).

Table3: prevalence of ferritin deficiency

	No	Percentage %
Normal (15 _150ng/ml)	78	61.9
Low (>15 150ng/ml)	48	38.1

Table 3 shows that 38.1% of the pregnant women had low ferritin levels (<15 ng/mL), indicating iron deficiency, while 61.9% had ferritin levels within the normal range (15–150 ng/mL).

Table 4: Prevalence of hemoglobin (Hb) levels

	No	Percentage %
Normal (11.5_15.5g/dL)	**11**	**8.7**
Low (<11.55g/dL)	**115**	**91.3**

The data indicate a high prevalence of anemia among the pregnant women in the study. Only 8.7% had normal hemoglobin levels (11.5– 15.5g/dL), while the majority—91.3%—had hemoglobin concentrations below 11.5 g/dL, consistent with anemia (Table 4).

Correlation between ferritin and Vitamin D levels

Table 5: Correlation between ferritin and Vitamin D levels in Pregnant Women

	Mean ± SD	R	P- value
Ferritin level	24.09±13.449	0.465**	0.001
Vitamin D	16.588±19.564		

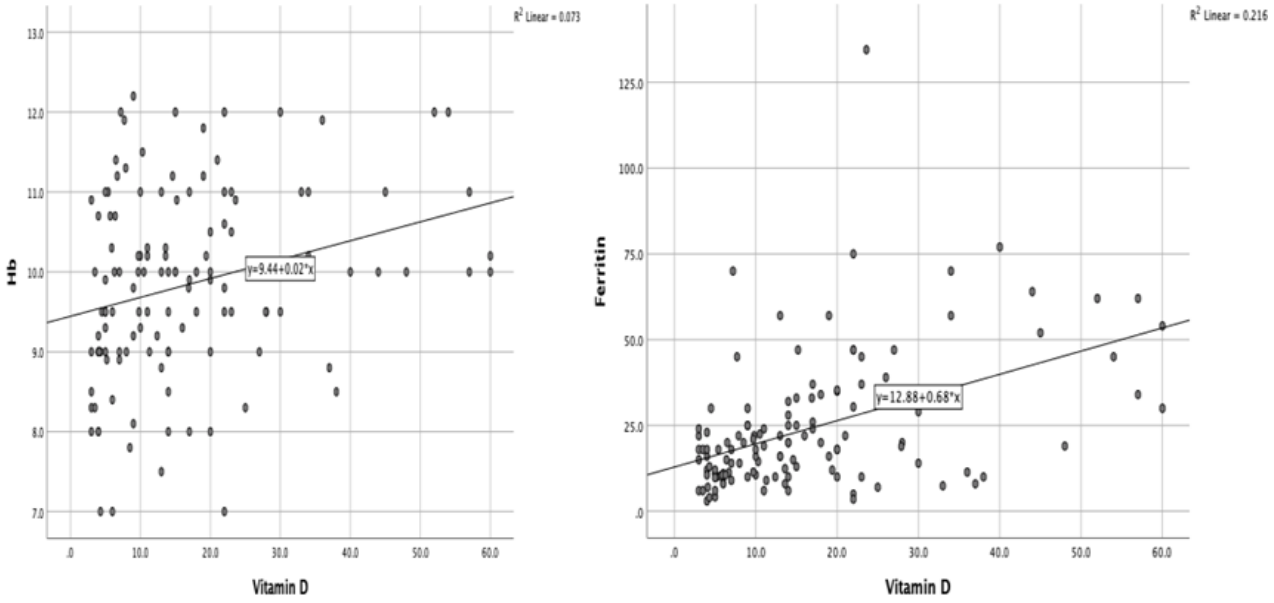
A statistically significant positive correlation was found between ferritin and vitamin D levels among the pregnant women ($r = 0.465$, $p = 0.001$). This suggests that higher vitamin D levels are moderately associated with higher iron stores.

Correlation Between Hemoglobin (Hb) and Vitamin D Levels

Table 6: Correlation Between Hemoglobin (Hb) and Vitamin D Levels in Pregnant Women

	Mean ± SD	R	P- value
Hb	9.837±1.180	0.269**	0.002
Vitamine	16.588±19.56		

A statistically significant but weak positive correlation was observed between hemoglobin and vitamin D levels (r = 0.269, p = 0.002). This suggests that pregnant women with higher vitamin D levels tend to have slightly higher hemoglobin concentrations.



6a: correlation between Hemoglobin and Vitamin D 6b: correlation between Ferritin and Vitamin D

Figure6: scatter plot for vitamin D compared to various parameters
6a:vitamin D and Hemoglobin showing a positive linear correlation
6b: vitamin D and Ferritin showing a positive linear correlation

DISCUSSION

The study, conducted among a cohort of pregnant women in Derna, Libya, highlights the potential relationship between vitamin D and iron deficiency anemia. In total, only 126 cases were submitted for analysis. Besides the prevalence of iron deficiency anemia in vitamin D-deficient patients, iron deficiency anemia itself may predispose patients to vitamin D deficiency, as those with anemia are less likely to receive adequate sunlight exposure upon admission to the hospital. Sunlight exposure is a natural source of vitamin D. (Basutkar RS et al 2019) The results of our study indicate a strong, statistically significant, positive correlation between vitamin D. levels and iron stores (ferritin and hemoglobin) in pregnant women, supporting the hypothesis that vitamin D deficiency may contribute to anemia through mechanisms including hepcidin regulation and inhibition of inflammation affecting iron absorption. Sixty-nine percent of the sample was found to be severely vitamin D deficient, and 91.3% were anemic. Ferritin deficiency was recorded in 38.1% of participants. These findings underscore the importance of including vitamin D assessment in routine pregnancy checkups and providing nutritional and pharmacological intervention strategies to mitigate its negative effects on maternal and fetal health. , Azizi-Soleiman et al. concluded that there may be a relationship between vitamin D and iron levels. However, this correlation was only observed in cross-sectional studies, and the authors noted that interventional studies did not provide sufficient evidence to support this association. Consistent with the findings of this review, a systematic review and meta-analysis of seven observational studies from the United States, France, and three Asian countries published between 2010 and 2013 found that individuals with vitamin D deficiency had a 64% higher risk of anemia. Both vitamin D and iron deficiencies are prevalent during pregnancy. Vitamin D levels are inversely related to hepcidin levels and directly related to hemoglobin and iron concentrations. Hepcidin is a hormone that regulates systemic iron homeostasis, controlling plasma iron levels and its distribution in tissues. The regulation of hepcidin helps to prevent the release of iron from macrophages, reduces iron release from hepatocytes, and inhibits iron absorption by enterocytes. (Lima MS, et al 2022). A study in the Northern United States revealed that 29.2% of Black women and 54.1% of Black neonates were vitamin D deficient, while 45.6% of Black women and 46.8% of Black neonates were considered insufficient. In comparison, 5% of White women and 42.1% of White neonates were vitamin D deficient, and 9.7% of White women and 56.4% of White neonates were insufficient. Another study conducted in Boston found that 28% of women with serum 25(OH)D levels below 37.5 nmol/liter underwent cesarean sections, compared to only 14% of those with levels at or above 37.5 nmol/liter. Additionally, a population-based study in Sydney, Australia, indicated a significant increase in the risk of neonatal vitamin D deficiency associated with maternal deficiency (OR 17.2, 95% CI 8.8–34.3). Infants born to deficient mothers had lower average birth weights (mean ± SD: 3245 g ± 545) compared to those born to sufficient mothers (mean ± SD: 3453 g ± 555), with a statistically significant difference ($p < 0.001$). (Basutkar RS et al 2019). Furthermore , several studies did not provide measures of association for the relationship between vitamin D and anemia during pregnancy, nor did they supply enough data to calculate this relationship. This limitation hindered the ability to perform a broader analysis involving more studies. (Lima MS, et al 2022). The study has several limitations. Its cross-sectional design prevents us from establishing a causal link between vitamin D deficiency and anemia. Vitamin D levels were measured only once, which may not accurately reflect the status throughout pregnancy. Key biomarkers, such as transferrin receptor, CRP, IL-6, and hepcidin, were not assessed, limiting our insight into iron metabolism and inflammation. Additionally, the use of convenience sampling from a single geographical location may affect the generalizability of the results. Despite these constraints, the study highlights a strong correlation and calls for further research to explore causality and the benefits of intervention.

Conclusion

This study provides compelling evidence of a significant association between vitamin D deficiency and anemia among pregnant women in Derna, Libya. The findings demonstrated that a substantial proportion of the study population exhibited severe vitamin D deficiency and low hemoglobin levels, with a moderate to strong positive correlation between vitamin D status and both ferritin and hemoglobin concentrations. These results suggest that

vitamin D may play a vital role in iron metabolism and erythropoiesis, potentially through the regulation of hepcidin and inflammatory pathways.

Given the high prevalence of vitamin D deficiency and anemia observed in the study sample, routine screening for vitamin D levels during pregnancy is strongly recommended. Integrating vitamin D assessment and appropriate supplementation into prenatal care protocols could contribute to reducing the burden of anemia and improving maternal and fetal health outcomes.

However, the cross-sectional design limits the ability to establish causality, and further longitudinal and interventional studies are warranted. Future research should also include broader biomarkers—such as hepcidin, inflammatory cytokines, and transferrin receptors—to deepen the understanding of the underlying mechanisms linking vitamin D deficiency to anemia during pregnancy.

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