Vitamin D Deficiency During Pregnancy: Risk for Preeclampsia and Adverse Neonatal Outcome

HANAN F. MOHAMED, M.D.*; SHERIF MAGDY, M.D.* and MOHAMED ELMOHANDES, M.D.**
The Departments of Clinical Pathology* and Obstetrics & Gynecology**, El Galaa Teaching Hospital

**Abstract**

Preeclampsia is a pregnancy specific syndrome characterized by high blood pressure and proteinuria after 20 weeks gestation that may occur in up to 8% of pregnant women. Women who develop preeclampsia are at increased risk for development of pulmonary oedema, coagulation defects, hepatic or renal failure, seizures and even death. Infants born to preeclamptic mothers are at increased risk of prematurity and more likely to be small for gestational age.

An emerging area of study that has garnered significant attention, is the role of vitamin D in preeclampsia. Vitamin D has direct influence on the molecular pathways proposed to be important in the pathogenesis of preeclampsia.

Our objective was to assess the effect of maternal 25-hydroxy vitamin D deficiency on the risk of preeclampsia and to assess the vitamin D status of newborns of preeclamptic mothers.

Records were obtained for 160 pregnant women with singleton pregnancy followed from less than 16 weeks gestation till delivery. 30 subjects met the criteria for diagnosis of preeclampsia as described below and served as (cases group), 90 cases remained normotensive without any complications till delivery and served as (control group). The rest who developed gestational diabetes, autoimmune disorders or other medical diseases were excluded.

Serum 25 hydroxy vitamin D concentrations were measured by vidas kit on Mini VIDAS immunoassay analyzer supplied by Bio Merieux using the ELFA technique (Enzyme Linked Fluorescent Assay).

Our results revealed that Serum 25 hydroxy vitamin D was significantly decreased among patients with preeclampsia compared to healthy control subjects 18.1 ±5.4ng/ml vs 32.1±9.8ng/ml (p-value 0.0003).

Cord serum 25(OH)D concentration were significantly lower among neonates of preeclamptic mothers than among neonates of non preeclamptic control mothers (17.7±2.9ng/ml vs 21.6±2.3ng/ml, p=0.02).

Logistic regression analysis when performed, demonstrated that 25(OH)D at levels <20ng/ml was statistically significantly associated with a tendency towards increased risk of preeclampsia [Odds Ratio 3.8,95% CI (1.6-9.1), p 0.002]. A strong inverse relation between serum 25(OH)D and the probability risk of preeclampsia was derived from linear regression correlation curve (slope = -0.102, r²=0.877, p=0.0009).

In conclusion, our study reinforces the potential link between vitamin D deficiency and preeclampsia risk. Thus preeclampsia may be added to the growing list of adverse health consequences of maternal vitamin D deficiency. Supplementing vitamin D among deficient women either in the preconception period or in early pregnancy should be explored as a safe and effective means of preventing preeclampsia and promoting neonatal well-being.

Thus, we recommend early screening of all pregnant women for vitamin D deficiency for earlier supplementation to mitigate its adverse outcomes.

**Key Words:** Vitamin D deficiency – Adverse pregnancy outcome – Preeclampsia.

**Introduction**

PREGNANCY is a state of increased requirement of macro- and micro-nutrients, making the pregnant women susceptible to develop deficiencies of various micronutrients [1]. Complications of pregnancy and childbirth are the leading causes of disability and death among women of reproductive age in developing countries, accounting for at least 18% of the global burden of disease in this age-group [2]. Similarly, the pattern of leading causes of maternal death and disability are closely linked to high prevalence of vitamin D deficiency, poor maternal health during pregnancy, inadequate care during delivery, and lack of newborn care [3].

Vitamin D is a steroid hormone that is derived primarily from synthesis in the skin through exposure to ultraviolet B radiation. Vitamin D undergoes

---

**Correspondence to:** Dr. Hanan F. Mohamed, The Department of Clinical Pathology, El Galaa Teaching Hospital

**Abbreviations:**

25(OH)D : 25 hydroxy vitamin D.
CI : Confidence interval.
hydroxylation in the maternal liver to form 25 hydroxy vitamin D (25-OH-D) [4]. The active form of vitamin D (1,25-[OH]2-vitamin D) results from the activity of 1-a-hydroxylase in the maternal kidney or placenta. Because the half-life of 1,25-(OH)2-vitamin D is only several minutes, the more accurate assessment of an individual’s vitamin D status is determined through measurement of 25-hydroxy vitamin D [5].

Vitamin D has now been shown to be of considerable importance not only for bone health, but also for glucose regulation, immune function and good uterine contractility in labour [6].

Maternal vitamin D deficiency is a widespread public health problem. This vitamin D deficiency epidemic during pregnancy is caused by a lack of adequate sunlight exposure needed to synthesize vitamin D3 (cholecalciferol) in the skin, coupled with oral intakes that are too low to meet the increased demands of pregnancy [7].

The vitamin D stores in the infant start with transplacental transfer of 25(OH)D in early pregnancy from mother to fetus. It is very obvious that maintaining optimum vitamin D nutrition during pregnancy is essential for prevention of hypovitaminosis D in the fetus and vitamin D deficiency at birth and in early infancy [2]. Despite the reported high prevalence of deficiency and the possible consequences, the desired optimal level needed for pregnant women in their body and the amount of vitamin D intake required to maintain adequate levels is not very well documented [11].

Vitamin D deficiency during pregnancy has been linked with a number of serious short- and long-term health problems in offspring, including impaired growth, skeletal problems, type I diabetes, asthma, and schizophrenia [8].

Few investigators have explored the role of maternal vitamin D status in infertility, gestational diabetes mellitus, bacterial vaginosis and preeclampsia, however, the exact role and metabolism of vitamin D is not well understood [2].

Preeclampsia is a relatively common health condition among pregnant women, affecting 3-10% of pregnancies worldwide [7]. Preeclampsia has been described as a 2-stage disease in which stage I is heralded by poor placental invasion, development, and remodeling. Stage II develops later and involves the clinical recognition of preeclampsia in the form of maternal hypertension, proteinuria, and end-organ disease [9]. Women with preeclampsia may experience swelling of the face and hands, headache, vision changes, nausea and sudden weight gain [10]. If preeclampsia is left untreated, it can develop into eclampsia, a life threatening occurrence of seizures which is associated with multiple maternal and fetal adverse effects [11]. In addition, it has been reported that women with a history of preeclampsia are at elevated risk for cardiovascular disease later in life [19].

Preeclampsia starts after the 20th week of a pregnancy and up to 12 weeks after delivery [2]. Data that suggest an association between preeclampsia and vitamin D deficiency are developing contributing the pathogenesis of preeclampsia to a number of biological processes that may be directly or indirectly affected by vitamin D, including immune dysfunction, placental implantation, abnormal angiogenesis and excessive inflammation [11].

**Aim of the study:**

Our present study aimed to assess the effect of maternal 25-hydroxy vitamin D (25[OH]D) deficiency on the risk of preeclampsia before the onset of clinical symptoms and to assess the vitamin D status of newborns of preeclamptic mothers.

**Subjects and Methods**

Records of one hundred and sixty women with singleton pregnancy at less than 16 weeks gestation attending the Outpatient Clinic at El Galaa Teaching Maternity Hospital for Antenatal Care (aged from 20 to 38 years) were obtained in the period from April 2013 to February 2014 after a written consent was taken from each participant.

Patients with pregestational diabetes, chronic hypertension, multifetal gestation or any preexisting chronic medical disorders were excluded from the study.

Participants were subjected to:

- Full demographic and medical history
- 7ml blood was collected from each participant, 2ml on EDTA tube to perform complete blood count on sysmex (supplied by BM-Egypt), and 5ml on plain tube to perform glucose levels, liver and kidney functions on Hitachi 912 (supplied by Roche) and the rest of sera were stored at-70ºc until assay of vitamin D.
- All participants were followed-up clinically and laboratory till delivery with records of blood pressures and urinary protein measurements.
**Preeclampsia was diagnosed when:** (1) A pregnant woman developed gestational hypertension associated with proteinuria and return of all abnormalities to normal by 12 weeks postpartum. Gestational hypertension was defined according to WHO criteria as systolic blood pressure $\geq 140$ mmHg and/or diastolic blood pressure $\geq 90$ mmHg for the first time after 20 weeks’ gestation in an individual with previously normal blood pressure. Proteinuria was defined as the excretion of more than 300mg protein in 24h, a random sample of 2+ protein, or a protein-creatinine ratio more than 300mg/gmcreatinine. OR (2) The new development of decreased blood platelet (platelet count $<100,000$/microliter) with evidence of impaired kidney and liver functions.

30 subjects who met one or more of the above criteria for diagnosis of preeclampsia served as (cases group), 90 cases remained normotensive without any complications till delivery and served as (control group). The rest who developed gestational diabetes, autoimmune disorders or other medical diseases were excluded.

- Venous umbilical cord blood samples were collected from both cases and controls processed and sera were stored at $-70^\circ$C until assay of 25 hydroxy vitamin D.
- Delivery data were collected for both cases and control patients that included gestational age at delivery and assessment of intrauterine growth restriction that was based on $<10^{th}$ percentile birthweight, as assessed by gestational age of delivery.

Determination of 25 hydroxy vitamin D concentrations by vidas kit on Mini VIDAS immunoassay analyzer supplied by Bio Merieux using the ELFA technique (Enzyme Linked Fluorescent Assay) according to the manufacturers, instructions (E-69280 Marcy L, Etoile-France).

The assay principle combines an enzyme immunoassay competition method with a final fluorescent detection.

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. 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 450nm. 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.

**Statistical analysis:**

Data analysis was performed using SPSS for windows version 10. Results were reported as mean±standard deviation or as percentages. $p$-values $<0.05$ was considered significant. Regression analysis was conducted to estimate the effect of serum 25 hydroxy vitamin D concentration on the risk of preeclampsia.

Probability risk for preeclampsia was calculated from log odds using inverse logit function, probability risk=$e^{(\log odds)}/(1+e^{(\log odds)})$, where $e$ is the base of the natural log.

**Results**

Thirty patients with preeclampsia and ninety matched control subjects were included in this study.

Vitamin D status was reported for both preeclampsia and control groups.

We used cut off levels according to ACOG (American college of obstetricians and gynecologists) for definition of vitamin D deficiency. The same cutoffs were used for both women and neonates because the definition of vitamin D sufficiency does not vary by age [7,11,14]. (Normal, $>32$ng/mL; insufficient, $\geq 20$ and $\leq 32$ng/mL; and deficient, $<20$ng/ml).

Pregnancy demographic and outcomes for preeclamptic and control group are summarized in Table (1). Patients with preeclampsia were noted to be with greater body mass index than control group. As expected, cases of preeclampsia were delivered preterm because of their disease with gestational age at delivery $29.6 \pm 1.2$ weeks) compared to ($38.7 \pm 1.1$ weeks) in control group).

The incidence of intrauterine growth restriction in the preeclamptic group were significantly greater compared with healthy control subjects ($33\%$ versus $11\%, p<0.01$).
Serum 25 hydroxy vitamin D was significantly decreased among patients with preeclampsia compared to healthy control subjects 18.1 ± 5.4ng/ml vs 32.1±9.8ng/ml (p-value 0.0003) (Table 2, Fig. 1).

There was a statistically significant difference between preeclamptic group versus control group as regards vitamin D deficiency <20ng/ml (Table 3).

Cord serum 25(OH)D concentration were significantly lower among neonates of preeclamptic mothers than among neonates of non preeclamptic control mothers (17.7±2.9ng/ml vs 21.6±2.3ng/ml, p=0.02) (Table 4, Fig. 2).

A statistically significant difference was observed between groups of neonates of preeclamptic mothers versus neonates of control group as regards vitamin D deficiency <20ng/ml (Table 5).

To assess the effect of maternal serum 25(OH)D on the odds of having a diagnosis of preeclampsia, logistic regression analysis was performed and demonstrated that 25(OH)D at levels <20ng/ml was statistically significantly associated with a tendency towards increased risk of preeclampsia (Odds Ratio 3.8,95% CI (1.6-9.1), p 0.002) (Table 6).

A strong inverse relation between serum 25(OH)D and the probability risk of preeclampsia was derived from linear regression correlation curve (slope = −0.102, r²=0.877, p=0.0009) (Fig. 3).

Table (1): Demographic and outcomes of pregnancies complicated by preeclampsia (cases group) versus non preeclampsia (control subjects).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preeclampsia (cases)</th>
<th>Non preeclampsia (control)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age years (mean±SD)</td>
<td>27.7±6.5</td>
<td>28.1±4.8</td>
<td>0.8</td>
</tr>
<tr>
<td>Body mass index kg/m² (mean±SD)</td>
<td>32.1±3.3</td>
<td>26.2±2.3</td>
<td>0.004</td>
</tr>
<tr>
<td>Gestational age at blood collection, weeks</td>
<td>11.4±1.4</td>
<td>11.7±1.5</td>
<td>0.67</td>
</tr>
<tr>
<td>% Nulliparous</td>
<td>67%</td>
<td>56%</td>
<td>0.095</td>
</tr>
<tr>
<td>Gestational age at delivery, weeks (mean±SD)</td>
<td>29.6±1.2</td>
<td>38.7±1.1</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>% of intrauterine growth restriction</td>
<td>33%</td>
<td>11%</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Table (2): Vitamin D status in preeclamptic group versus control group.

<table>
<thead>
<tr>
<th>Vitamin D status</th>
<th>Preeclamptic women (cases group)</th>
<th>Non preeclamptic group (control group)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum 25(OH)D ng/ml</td>
<td>18.1±5.4</td>
<td>32.1±9.8</td>
<td>0.0003</td>
</tr>
<tr>
<td>Vitamin D status:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>7 (23%)</td>
<td>42 (47%)</td>
<td></td>
</tr>
<tr>
<td>25(OH)D &gt;32ng/ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insufficient</td>
<td>6 (20%)</td>
<td>25 (28%)</td>
<td></td>
</tr>
<tr>
<td>25(OH)D ≥20-32ng/ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficient</td>
<td>17 (57%)</td>
<td>23 (25%)</td>
<td>0.005</td>
</tr>
<tr>
<td>25(OH)D &lt;20ng/ml</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table (3): Comparison of vitamin D deficient and non deficient preeclamptic group versus control group.

<table>
<thead>
<tr>
<th>Vitamin D status</th>
<th>Preeclamptic group</th>
<th>Control group</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficient &lt;20ng/ml</td>
<td>17 (57%)</td>
<td>23 (25%)</td>
<td>0.005</td>
</tr>
<tr>
<td>Non deficient ≥20ng/ml</td>
<td>13 (43%)</td>
<td>67 (75%)</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Data are n (%).

Table (4): Vitamin D status in neonates of preeclamptic mothers versus neonates of non preeclamptic mothers.

<table>
<thead>
<tr>
<th>Vitamin D status</th>
<th>Neonates of preeclamptic mothers</th>
<th>Neonates of non preeclamptic mothers</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cord serum 25(OH)D ng/ml</td>
<td>17.7±2.9</td>
<td>21.6±2.3</td>
<td>0.02</td>
</tr>
<tr>
<td>Vitamin D status:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>6 (20%)</td>
<td>31 (34%)</td>
<td></td>
</tr>
<tr>
<td>25(OH)D &gt;32ng/ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Insufficient</td>
<td>13 (43.4%)</td>
<td>41 (46%)</td>
<td></td>
</tr>
<tr>
<td>25(OH)D ≥20-32ng/ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficient</td>
<td>11 (36.6%)</td>
<td>18 (20%)</td>
<td>0.04</td>
</tr>
<tr>
<td>25(OH)D &lt;20ng/ml</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table (5): Comparison of vitamin D deficient and non deficient neonates of preeclamptic mothers versus neonates of non preeclamptic mothers.

<table>
<thead>
<tr>
<th>Vitamin D status</th>
<th>Neonates of preeclamptic mothers</th>
<th>Neonates of non preeclamptic mothers</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficient &lt;20ng/ml</td>
<td>11 (36.6%)</td>
<td>18 (20%)</td>
<td>0.04</td>
</tr>
<tr>
<td>Non deficient ≥20ng/ml</td>
<td>19 (63.4%)</td>
<td>72 (80%)</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Data are n (%).
Table (6): Logistic regression analysis for preeclampsia risk according to vitamin D status.

<table>
<thead>
<tr>
<th>Serum 25 hydroxy vitamin D</th>
<th>Control group (n)</th>
<th>Preeclampsia group (n)</th>
<th>Odds Ratio (95%CI)</th>
<th>p-value</th>
<th>Relative risk</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deficient 25(OH)D &lt;20ng/ml</td>
<td>23</td>
<td>17</td>
<td>3.8 (1.6-9.1)</td>
<td>0.002</td>
<td>2.2</td>
<td>0.0009</td>
</tr>
<tr>
<td>Insufficient 25 (OH)D 20-32ng/ml</td>
<td>25</td>
<td>6</td>
<td>4.4 (0.43-4.76)</td>
<td>0.55</td>
<td>1.23</td>
<td>0.49</td>
</tr>
</tbody>
</table>

Discussion

Vitamin D deficiency has been associated with several adverse pregnancy outcomes and is a public health issue worldwide. Epidemiologic data suggests that vitamin D deficiency may be involved in the pathophysiologic condition of preeclampsia.

In this study, we examined serum 25(OH)D among those patients who subsequently experienced preeclampsia after 20 weeks of gestation. We found a significantly decreased level of 25(OH)D in those patients (cases group) when compared to control group who remained normotensive till delivery and 12 weeks postpartum (18.1 ± 5.4ng/ml versus 32.1 ± 9.8ng/ml respectively, p=0.0003). An inverse relationship exist between serum 25(OH)D and the probability risk of preeclampsia (slope = –0.102, \( r^2 =0.877, p=0.0009 \)) We also demonstrated that 25(OH)D at levels <20ng/ml was statistically significantly associated with a tendency towards increased risk of preeclampsia [Odds Ratio 3.8,95% CI (1.6-9.1), p 0.002].

In consistent with our study, Baker et al., [12], in a nested case control study showed that vitamin D deficiency was associated with an increased risk of preeclampsia, they found that Midgestation 25(OH)D levels were lower in women who developed severe preeclampsia compared to uncomplicated pregnancies (p<0.01). It was also seen that levels of 25(OH)D less than 16ng/ml were associated with a significant odds of 5 fold increased risk compared with levels of at least 24ng/ml (odds ratio 5.41,95% CI (2.02-14.5).

A similar study conducted by Bodnar et al., [13], on singleton pregnant women followed from 16 weeks gestation till delivery. They found that 25(OH)D levels were significantly lower in cases (preeclamptic women) when compared to controls. They also found a monotonic dose response relation between serum 25(OH)D concentrations and risk of preeclampsia and that a 18ng/ml decline in 25(OH)D doubled the risk of preeclampsia.

Similarly also, Robinson et al., [14], demonstrated a significantly decreased levels of 25(OH)D in patients with early onset severe preeclampsia relative to healthy control pregnancies that were...
matched for gestational age at sampling (median 25(OH)D 18ng/ml versus 32ng/ml \( p < 0.001 \)). They also found that at plasma levels of 25(OH)D \( \leq 19.6 \)ng/ml, there was a 3.6 fold increased odds of diagnosis of early onset severe pre-eclampsia (odds ratio 3.60, 95% CI 1.71-7.58 \( p < 0.001 \)).

Evidence that vitamin D supplementation may reduce the risk of pre-eclampsia was provided by Haugen et al., [16] in a prospective cohort study from Norway on 23,423 nulliparous pregnant women looking at the use of vitamin D supplements during pregnancy. The study demonstrated that vitamin D supplements reduced the risk of pre-eclampsia by 27% (odds ratio = 0.73; 95% CI: 0.58 –0.92). It also showed that a total dietary intake of vitamin D of 15-20 microg/d was associated with a significant 24% reduction in risk of pre-eclampsia compared to less than 5 microg/d. Therefore, overall there is a very strong evidence based on epidemiological studies of a link between vitamin D deficiency and risk of pre-eclampsia in pregnant women.

In contrast to our study, Powe et al., [15] conducted another nested case control study and compared first trimester total and free 25(OH)D levels among women who subsequently developed pre-eclampsia versus normotensive pregnancies showing the levels to be similar in cases and controls (27.4 ± 1.9 versus 28.8 ± 0.8ng/ml \( p =0.43 \)). They found a tendency towards increased risk of pre-eclampsia with 25(OH)D levels \( <15 \)ng/ml, but the association was not significant (odds Ratio 1.35, 95% CI (0.40-4.50).

Many authors have proposed the biologically plausible mechanisms by which maternal vitamin D status could alter risk of pre-eclampsia. 25(OH)D deficiency was reported to be associated with inflammation-linked vascular endothelial dysfunction [23]. The active form of vitamin D, 1,25-dihydroxy vitamin D \( \Delta_3 \), has been shown to regulate the transcription and function of genes associated with placental invasion, normal implantation, and angiogenesis [11,17]. Furthermore, abnormal implantation is proposed to be mediated at least in part by an inappropriate immune response between mother and baby. The immunomodulatory properties of 1,25-dihydroxy vitamin D \( \Delta_3 \) may be relevant in this regard [18,23]. Maternal vitamin D deficiency may likewise predispose to the increased inflammatory response. Notably, vascular structure and function including vascular compliance, elasticity, and intima media thickness [11,19]. Vitamin D deficiency may also elevate blood pressure through its negative endocrine regulator effect on renin angiotensin [20]. Finally, the proteinuria of pre-eclampsia is thought to be mediated by renal vascular endothelial growth factor (VEGF). 1,25-Dihydroxy vitamin D \( \Delta_3 \) has been shown to regulate angiogenic processes through direct effects on VEGF gene transcription [11,21].

Our results highlight that neonates of preeclamptic mothers are at particularly high risk of vitamin D deficiency when compared to neonates of non-preeclamptic group (36.6% versus 20%, \( p =0.04 \)). However, these results are not unexpected, the fetus relies entirely on vitamin D stores of the mother, so if the mother is deficient so is the fetus [22]. Similar to our findings, Bodnar et al., [18], observed that neonates born to preeclamptic mothers were significantly more likely to have poor vitamin D status than neonates of control mothers. Mean 25(OH)D of Cord serum in neonates of preeclamptic pregnancies versus neonates of non-preeclamptic pregnancies [39.2 (95\% CI 32.1-47.9) vs 50.3 (95\% CI 43.6-58.1] respectively \( p=0.001 \). Because early-life vitamin D deficiency has been associated with adverse health outcomes in off spring, neonates of preeclamptic mothers may be an easily identified group that needs a targeted intervention to improve vitamin D status from birth.

Some limitations in the current study that should be noted and addressed in future investigations: There was no assessment of baseline dietary vitamin D intake or sun exposure in the study population.

In conclusion, our study reinforces the potential link between vitamin D deficiency and preeclampsia risk. If our results are further confirmed by others, they suggest that preeclampsia may be added to the growing list of adverse health consequences of maternal vitamin D deficiency. Supplementing vitamin D among deficient women either in the preconception period or in early pregnancy should be explored as a safe and effective means of preventing pre-eclampsia and promoting neonatal well-being.

However, follow-up studies and additional research will be necessary to tease out further details of this 25(OH)D preeclampsia relationship to understand the impact of this vitamin deficiency on adverse pregnancy consequences. As vitamin D deficiency is clinically silent until severe events occur, our recommendation allows for early screening of all pregnant women for vitamin D deficiency for earlier supplementation to mitigate its adverse outcomes.
References


