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Original article

The effect of cortisol on pain intensity in adolescent girls with primary dysmenorrhea: A double-blind, randomized, placebo-controlled trial

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Abstract

Dysmenorrhea is a prevalent gynecological condition affecting women of reproductive age. Primary dysmenorrhea (PD) typically begins during adolescence. PD is associated with varied intensities of discomfort, leading to both physical and emotional distress. Elevated cortisol secretion impacts the reproductive system and contributes to the occurrence of pain and autonomic symptoms during menstruation.

Objective: To determine the impact of vitamin D supplementation on menstrual pain and its association with salivary cortisol in adolescents with PD.

Methods: A total of 191 adolescent females with primary dysmenorrhea were randomized into two groups: the study group (n=96) received vitamin D (4000 IU), while the control group (n=95) received a placebo for three months. Before and after the intervention, salivary cortisol levels were measured four times: in the morning, afternoon, evening, and night. The Visual Analogue Scale was used to assess the level of pain.

Results: Group baseline characteristics were similar. Compared to the placebo, vitamin D led to a significant decrease in mean VAS from 6.0 ± 1.8 to 2.49 ± 1.2 after 12 weeks ($p<0.001$). Both groups exhibited mild declines in cortisol levels, which were not statistically significant. Vitamin D arm revealed positive correlations between post-intervention pain and morning ($r = 0.34$), evening ($r = 0.51$), and night ($r = 0.38$) cortisol (all $p \leq 0.002$), while placebo showed only a modest association ($r = 0.25$, $p = 0.034$). There were no major complications.

Conclusion. Vitamin D markedly reduces menstrual pain in adolescents, while systemic cortisol changes are minimal. Nonetheless, residual pain remains linked to diurnal cortisol, suggesting that combining vitamin D with stress-modulating strategies may yield superior analgesia for PD.

Keywords: vitamin D, cortisol, dysmenorrhea, adolescent girls, pain.

1. Introduction

Primary dysmenorrhea (PD), characterized by painful menstruation without pelvic pathology, is a highly prevalent gynecological issue among adolescents, with self-reported incidence rates between 60% and 90% globally [1-3]. In Central Asia, as many as 78% of school-aged girls experience absenteeism or diminished academic performance due to menstrual pain, highlighting its socio-economic impact. In addition to acute discomfort, PD correlates with diminished health-related quality of life, increased anxiety and depressive symptoms, and prolonged central sensitization that may predispose individuals to chronic pain disorders [4-6].

The established pathophysiological model indicates that excessive endometrial production of prostaglandins results in myometrial hypercontractility, uterine ischemia, and activation of nociceptors [1,7,8]. In accordance with this perspective, initial treatment focuses on non-steroidal anti-inflammatory drugs (NSAIDs) and combined oral contraceptives (COCs) [9, 10]. However, up to one-third of individuals achieve insufficient relief or encounter adverse effects, necessitating the investigation of adjunctive or alternative therapies [2,3,11].

Increasing evidence suggests that dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis and its effector hormone cortisol may serve as a concurrent mechanism contributing to menstrual pain. Cross-sectional studies indicate increased salivary or serum cortisol levels in adolescents with pain disorders (PD) compared to pain-free controls, with higher concentrations positively correlating with Visual Analogue Scale (VAS) scores [12,13]. Experimental data indicate that prolonged menstrual pain is associated with enhanced central pain processing and an expanded pain distribution, significantly influenced by stress hormones [6,14]. Neuroimaging studies further indicate that modifications in the cognitive control network facilitate the relationship between extended dysmenorrheic pain and anxiety symptoms, again implicating cortisol-related pathways [15]. Interventions that mitigate stress or regulate cortisol dynamics-such as endorphin massage, low-level laser therapy, pulsed electromagnetic field exposure, and peppermint aromatherapy-concurrently decrease cortisol levels and perceived pain intensity [13,16,17]. These convergent findings establish cortisol as both a biomarker and a potentially alterable target in Parkinson's disease.

Notwithstanding these insights, the causal role of cortisol in nociceptive experience in Parkinson's disease remains ambiguous. Previous studies were predominantly observational, involved limited sample sizes, or lacked stringent blinding and placebo controls. Furthermore, no randomized controlled trial (RCT) has investigated whether acute pharmacological intervention

of cortisol during menstruation directly alleviates pain in adolescent females. Addressing this gap is clinically significant due to the adolescent-specific interaction among endocrine development, stress responsiveness, and menstrual regulation [18-20].

This double-blind randomized placebo-controlled trial was designed to assess the impact of pharmacological cortisol suppression on pain intensity in adolescent girls with PD. We posited that temporary suppression of cortisol during the initial two days of menstruation would yield a clinically significant decrease in VAS pain scores relative to placebo. Secondary objectives encompassed evaluating alterations in pain-related functional impairment, mood variables, and salivary cortisol dynamics during the early follicular phase. This study seeks to elucidate the mechanistic role of cortisol in Parkinson's disease, with the objective of guiding the creation of targeted, mechanism-based therapies that enhance current prostaglandin-focused treatments and ultimately elevate the quality of life for impacted adolescents.

2. Materials and Methods

The study utilized a double-blind, randomized, placebo-controlled trial methodology.

Initially, 232 participants were enrolled prior to the commencement of the study. A total of 191 adolescent girls with primary dysmenorrhea (PD) participated in the study. We removed the remaining 41 participants for several reasons, including failure to meet inclusion criteria or withdrawal of consent. Participants were randomly allocated to two equivalent groups using a random number method: the study group (n=96), which received vitamin D (4000 IU, tablets produced in Poland), and the control group (n=95), which received placebo tablets, for a period of three months. The drugs were packaged and labeled by an independent expert, ensuring that neither the participants nor the researchers could discern which group received which preparation. The researchers acquired access to the participant identification numbers and the associated treatment forms solely after the completion of data collection.

At the end of the investigation, 168 girls (87 from the study group and 81 from the control group) completed the trial. Participants were excluded for reasons of consent withdrawal, illness, or relocation [18].

The research was executed following a standardized methodology at the outpatient section of the Regional Perinatal Center, overseen by a pediatric and adolescent gynecologist. Data were collected on complaints and detailed medical history, anthropometric measurements (weight, height, body mass index (BMI)) were documented, and pain intensity was assessed using the Visual Analog Scale (VAS). Salivary cortisol concentrations were assessed at four intervals during the day: morning, afternoon, evening, and night (pre-bedtime). These evaluations were performed at baseline and after three months of vitamin D or placebo administration.

Non-invasive sampling of biological material was readily conducted among adolescents, since it circumvented the stress linked to venous blood collection and laboratory visits. At the outset of the trial, all adolescent participants and their parents were comprehensively briefed on the study's objectives and methods. Informed consent was secured from each participant, highlighting the voluntary aspect of involvement and the right to withdraw at any point throughout the study.

The study results were statistically analyzed using SPSS version 26 (IBM SPSS Statistics, USA). The Wilcoxon signed-rank test for paired data and the Mann–Whitney U test were used to compare quantitative variables between groups. ANOVA was employed to evaluate repeated assessments of cortisol levels between the two groups pre- and post-intervention. The Spearman correlation test was employed to ascertain relationships between variables. All data are expressed as frequency percentages and mean ± standard deviation (SD), with $p<0.05$ being statistically significant.

3. Results

A total of 168 adolescent girls with primary dysmenorrhea were analyzed. The study and control groups were the same and had no significant differences in their starting characteristics, such as average age (14.16 ± 1.18 years), height (161.55 ± 6.05 cm), weight (51.4 ± 8.5 kg), BMI (19.7 ± 3.1 kg/m²), and pain intensity level measured by the Visual Analog Scale (VAS) (5.9 ± 1.9 points).

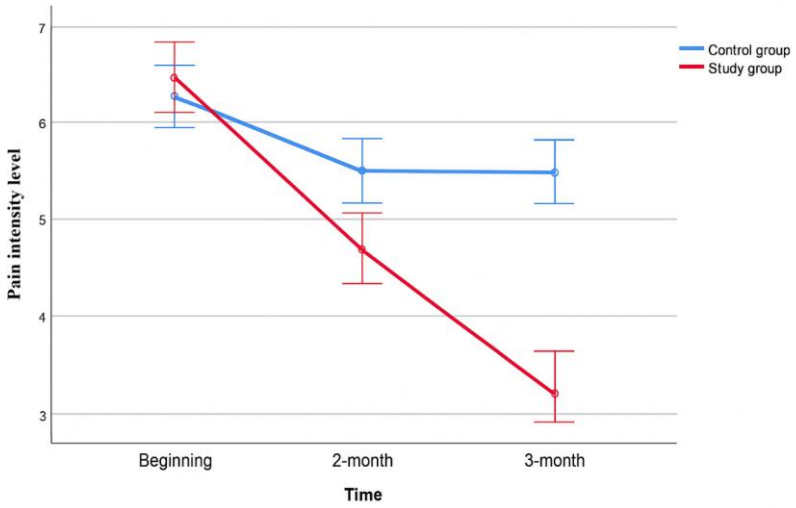


Figure 1 - Pain intensity level according to the VAS in the study and control groups before and after the intervention

Figure 1 illustrates a decline in pain intensity, as measured by the VAS, in both groups following three months of intervention. The intervention group exhibited a significant reduction in the mean VAS score, decreasing from 6.0 ± 1.8 to 2.49 ± 1.2. In contrast, the control group showed a decrease from 5.7 ± 1.9 to 5.0 ± 2.3. A statistically significant decrease in pain intensity was noted in the intervention group (p=0.00), while no significant changes were observed in the control group

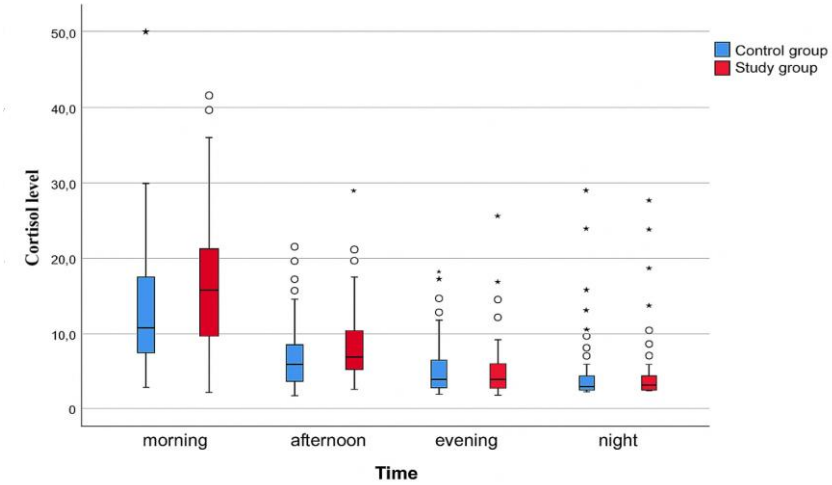
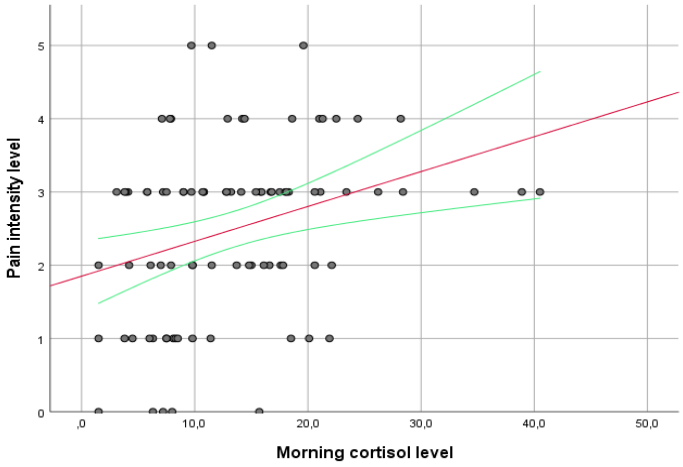


Figure 2 - Cortisol levels in the study and control groups after the intervention

Prior to the intervention, there was no significant difference in mean cortisol levels between the study and control groups. Following three months of vitamin D and placebo administration, a decline in salivary cortisol levels was noted in both groups; however, the differences were not statistically significant (Figure 2).



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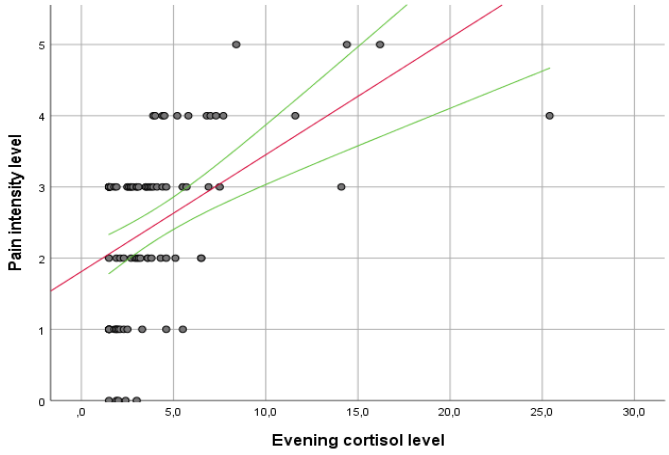

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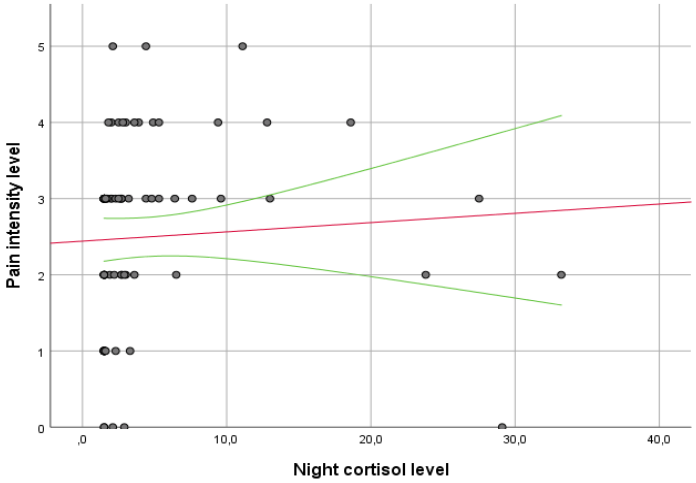

C

Figure 3 - Correlation between pain intensity and morning (A), evening (B) and night (C) cortisol levels in the study group

A weak positive association was found between the intensity of pain and the levels of salivary cortisol throughout the daytime on the part of adolescent girls who were diagnosed with primary dysmenorrhea (r = 0.167, p = 0.036). This correlation was seen prior to the beginning of the study.

In the study group, after the intervention, moderate positive correlations were identified between pain intensity and morning cortisol levels (r = 0.34, p = 0.002) (Figure 3a), evening cortisol levels (r = 0.51, p = 0.000) (Figure 3b), as well as nighttime salivary cortisol levels (r = 0.38, p = 0.000) (Figure 3c).

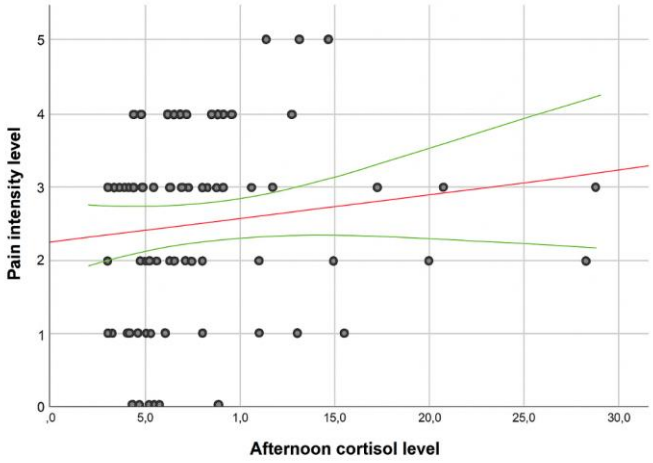

Figure 4 - Correlation between pain intensity and afternoon cortisol levels in the control group

After the intervention, there was a weak positive association found between the intensity of the pain and the levels of cortisol in the afternoon in the control group (r = 0.252, p = 0.034) (Figure 4).

4. Discussion

The present double-blind randomized trial demonstrates that three months of vitamin D supplementation produced a clinically meaningful 58 % reduction in menstrual pain intensity in adolescents with primary dysmenorrhea (PD), whereas placebo yielded only a marginal (12 %) and non-significant change. These results concur with earlier randomized studies showing that vitamin D decreases either VAS scores, analgesic consumption, or both in PD by 30–70 % [26–30]. The level of improvement we saw (about 3.5 points on the VAS) qualifies as "much improved" on the Patient Global Impression of Change scale, highlighting its real-world importance.

Several mechanisms may explain this antinociceptive effect. Vitamin D reduces the production of COX-2 and prostaglandins in endometrial stromal cells, which helps lessen excessive contractions of the uterus that cause pain during periods. It also helps the myometrium manage calcium levels better, leading to more organized and less painful contractions. Less explored, but increasingly compelling, is vitamin D's ability to modulate the hypothalamic-pituitary-adrenal (HPA) axis: serum 25-hydroxyvitamin D inversely correlates with basal and stress-induced cortisol, and supplementation can attenuate cortisol surges during inflammatory or psychological stressors [18, 21].

In our cohort, however, mean salivary cortisol declined modestly and non-significantly in both study arms. The absence of a between-group difference suggests that vitamin D's primary analgesic action occurred downstream or independent of systemic cortisol changes. This finding is not unprecedented; LAK et al. noted disproportionate pain relief relative to modest cortisol shifts after combined vitamin D + E therapy [22]. One explanation is that vitamin D's local anti-inflammatory effects in the uterus and central modulation of nociceptive pathways outweigh any modest systemic endocrine changes it triggers.

Notwithstanding the group-level stability in cortisol, post-intervention within-group analyses revealed moderate positive correlations between pain intensity and salivary cortisol at all three circadian time points in the intervention arm (r = 0.34–0.51). These connections support the idea that cortisol increases pain and are similar to earlier studies showing that higher daily cortisol levels are related to more pain from primary dysmenorrhea, anxiety, and increased sensitivity to pain. The strongest connection was found in the evening, which supports the idea that chronic pain affects the normal daily pattern of cortisol, resulting in a flatter curve that suggests more pain and negative feelings the next day.

Why then did vitamin D not significantly depress cortisol despite alleviating pain? One possibility is that our once-daily sampling underestimated nuanced changes in the diurnal rhythm; high-frequency or area-under-the-curve measures may be more sensitive [14]. Another reason could be that vitamin D improved the activity of vitamin D receptors in tissues, which reduced pain caused by prostaglandins, while ongoing stress from adolescence (like school pressure and body image issues) kept the HPA axis active. The weak but significant afternoon pain–cortisol link in the placebo group supports the premise that cortisol reactivity remains an independent pain modulator when prostaglandin pathways are pharmacologically untouched.

Clinically, our data argue for a two-pronged approach: nutrient or pharmacologic strategies that target prostaglandin metabolism (vitamin D, NSAIDs) and behavioral or physical interventions that normalize HPA tone—e.g., endorphin massage, Pilates, and low-level laser therapy—all shown to lower cortisol and pain in PD [16, 17, 23]. Future work could test synergistic combinations and explore whether evening-focused stress-reduction programs offer disproportionate benefit, given the prominent evening cortisol–pain coupling we identified.

These findings reinforce vitamin D supplementation as an inexpensive, well-tolerated adjunct for PD pain, even when systemic cortisol remains largely unchanged. They also highlight cortisol's continuing influence on residual pain, advocating adjunctive stress-modulating therapies. Prospective trials should incorporate high-resolution cortisol profiling, neuroimaging markers of central sensitization, and multi-modal interventions that combine anti-inflammatory and HPA-targeted components to achieve more complete analgesia for adolescent girls with PD.

5. Conclusions

Vitamin D supplementation for three months markedly alleviated menstruation pain in teenagers with primary dysmenorrhea, decreasing VAS scores by more than fifty percent, whereas the placebo exhibited minimal impact. The intensity of pain remained strongly correlated with cortisol levels, suggesting that stress-related HPA activity continues to affect residual pain. Consequently, vitamin D serves as a safe, economical supplement; nevertheless, its combination with stress-modulating techniques may produce superior analgesic effects for this population.

Conflicts of interest. None to declare.

Financing. None.

Author contributions. Conceptualization - A.A.; methodology – A.A., D.K.; writing (original draft preparation) – A.A., A.D., Sh.K.; writing (review and edition) – A.A. and D.K.

All authors have read, agreed to release version of a manuscript and signed the Author's right transfer form.

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Жасөспірім қыз балалардағы біріншілік дисменорея кезінде ауырсыну қарқындылығына кортизолдың әсері: Қос соқыр, рандомизацияланған, плацебо-бақыланатын зерттеу

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Түйіндеме

Дисменорея - репродуктивті жастағы әйелдерде жиі кездесетін гинекологиялық проблемалардың бірі. Біріншілік дисменорея әдетте жасөспірім кезінен басталады. Біріншілік дисменорея әртүрлі дәрежедегі ауырсынумен бірге жүреді. Бұл физикалық қана емес, сонымен қатар эмоционалды стрессті де тудырады. Кортизол секрециясының жоғарылауы репродуктивті жүйеге әсер етеді және етеккір кезінде ауырсыну мен вегетативті синдромдардың болуында маңызды рөл атқарады.

Зерттеудің мақсаты: D дәруменінің менструалдық ауырсынуға және сілекей кортизолымен байланысына әсерін бағалау.

Әдістері. Бастапқы дисменореясы бар 191 жасөспірім қыз екі топқа рандомизацияланды: үш ай бойы D дәрумені (4000 ХБ) қабылдаған негізгі (n=96) және плацебо қабылдаған бақылау (n=95). Сілекейдегі кортизол деңгейі зерттеуге дейін және кейін 4 уақыт аралығында (таңертең, түстен кейін, кешке, түнде) өлшенді. Ауырсыну қарқындылығы визуалды аналогтық шкала (ВАШ) бойынша бағаланды.

Нәтижесі. Бастапқы сипаттамалар топтар арасында ұқсас болды. Плацебомен салыстырғанда, D дәрумені 12 аптадан кейін орташа ВАШ көрсеткішін 6,0±1,8-ден 2,49±1,2-ге дейін айтарлықтай төмендетті ($p<0,001$). Екі топта да кортизол деңгейі шамалы төмендеді, бірақ бұл өзгеріс статистикалық мәнге жетпеді. Негізгі топта араласудан кейін ауырсыну деңгейі таңертеңгі ($r=0,34$), кешкі ($r=0,51$) және түнгі ($r=0,38$) кортизолмен оң корреляция көрсетті (барлық $p \leq 0,002$), ал бақылау тобында тек әлсіз байланыс байқалды ($r=0,25$; $p = 0,034$). Айтарлықтай асқынулар тіркелген жоқ.

Қорытынды. D дәрумені жасөспірімдердегі менструальды ауырсынуды айтарлықтай азайтады, ал жүйелі кортизол деңгейінің өзгерістері минималды. Дегенмен, қалдық ауырсыну тәуліктік кортизолмен байланысты болып қала береді, бұл D дәруменін стрессті реттейтін стратегиялармен біріктіру біріншілік дисменорея кезінде неғұрлым тиімді ауырсынуды басатын әсер беруі мүмкін екенін көрсетеді.

Түйін сөздер: D дәрумені, кортизол, дисменорея, жасөспірім қыздар, ауырсыну.

Влияние кортизола на интенсивность боли при первичной дисменорее у девочек-подростков: двойное слепое рандомизированное плацебо-контролируемое исследование

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Резюме

Дисменорея - одна из самых распространенных гинекологических проблем у женщин репродуктивного возраста. Первичная дисменорея обычно начинается в подростковом возрасте. Первичная дисменорея характеризуется различной степенью болевых ощущений и вызывает как физический, так и эмоциональный стресс. Увеличение секреции кортизола воздействует на репродуктивную систему и способствует возникновению болевого и вегетативного синдромов во время менструации.

Цель исследования: изучить влияние приема витамина D на уровень кортизола и интенсивность боли при первичной дисменорее у девочек-подростков.

Методы. 191 девочка-подросток с первичной дисменореей были рандомизированы на две группы: основную (n=96), получавшую витамин D (4000 МЕ) в течение трех месяцев, и контрольную (n=95), получавшую плацебо. Уровень кортизола в слюне измерялся четыре раза в сутки (утро, день, вечер, ночь) до и после вмешательства. Интенсивность боли оценивалась по визуально-аналоговой шкале.

Результаты. Исходные характеристики групп были сопоставимы. По сравнению с плацебо витамин D достоверно снизил средний балл по визуально-аналоговой шкале (ВАШ) с 6,0 ± 1,8 до 2,49 ± 1,2 через 12 недель ($p<0,001$). В обеих группах наблюдалось лёгкое, статистически недостоверное снижение уровня кортизола. В основной группе после вмешательства выявлены положительные корреляции между интенсивностью боли и утренним ($r=0,34$), вечерним ($r=0,51$) и ночным ($r=0,38$) кортизолом ($p\leq0,002$ для всех); в контрольной группе отмечена лишь умеренная связь ($r=0,25$; $p=0,034$). Серьёзных осложнений не зарегистрировано.

Выводы. Витамин D значительно снижает менструальную боль у подростков, при этом системные изменения уровня кортизола минимальны. Тем не менее, сохраняющаяся боль остаётся связанной с суточными колебаниями кортизола, что позволяет предположить, что сочетание витамина D с методами модуляции стресса может обеспечить более выраженный анальгезирующий эффект при первичной дисменорее.

Ключевые слова: витамин D, кортизол, дисменорея, девочки-подростки, боль.