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Association of Acupressure at Sanyinjiao Point 6 with C-Reactive Protein, Interleukin 6, Prostaglandin F2α, and Vasopressin in Medical College Students with Primary Dysmenorrhea.

Samira Amjad, Qamar Aziz, Ruqaya Nangrejo

ABSTRACT

Objective: To observe the changes in biochemical markers after applying acupressure in Primary Dysmenorrhea (PD) at Sanyinjiao Point 6 (SP6) and ascertain whether pain relief is biochemically mediated.

Study design and setting: A quasi-experimental design was adopted. The study was carried out from September 2022 to April 2023, in Karachi, Pakistan, at a private medical college.

Methodology: After ethical approval (BMU-EC/04-2022), 50 participants were selected using a modified menstrual symptom questionnaire, who were healthy, experienced regular PD, and abstained from analgesics. Written informed consent was taken, then in the first month of the study, blood samples (pre-intervention) were taken on day one of the menstrual cycle (MC). In the following three MCs, acupressure was self-applied at acupoint SP6, taught by a qualified acupressurist. During dysmenorrhea, it was applied on both legs, three times/day, for two to five minutes. In the third month of acupressure, second (post-intervention) blood samples were taken. Plasma levels of C-reactive protein (CRP), Interleukin-6 (IL-6), Prostaglandin F2α (PGF2α), and Vasopressin (VP) were measured and compared with pre-intervention samples using Statistical Package for Social Sciences 23 (SPSS 23).

Results: Median with interquartile ranges (IQR) of PGF2α, IL-6, VP, and CRP were obtained and compared, reporting a statistically significant increase in the post-intervention plasma CRP level (p=0.03), whereas plasma levels of PGF2α, IL-6, and VP showed no significant change (p=0.60, p=0.84, p=0.19, respectively).

Conclusions: Except CRP, the biochemical markers showed no significant change; hence, acupressure mediates its pain relief through additional mechanisms.

Keywords: Acupressure, C-Reactive Protein, Interleukin 6, Dysmenorrhea, Prostaglandins F, Vasopressins

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INTRODUCTION

Dysmenorrhea is pain during menstrual cycles (MC), and it is the most common gynecological problem found among women.[1] Without pelvic pathology, it is called primary dysmenorrhea (PD), and secondary if pathology is present.[2] PD starts with ovulatory cycles, often one year after menarche, but in some cases it may start after 2 years.[1] For some women, this pain is minimal and bearable, but for some it is so severe that it interferes with normal daily life.

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Dysmenorrhea is not reported as much as it actually prevails, nor is it adequately treated.[1] The prevalence in different countries; in Pakistan at 91.5%,[1] in Kuwait at 85.6%,[4] in Lebanon at 80.9%,[5] Spain at 47.8%,[4] and Poland at 94%.[1] Due to dysmenorrhea, in one year 58% of students missed at least one academic day, 13.9% missed one exam, 26% needed a clinical visit, and 4.1% were hospitalized.[4] In a Spanish study, 51.3% of dysmenorrhea sufferers claimed a lack of concentration, of which 47.8% had PD.[6]

Considering the risk factors, a study found that medical students had a greater chance of having PD than non-medical students, we can speculate that this may be due to increased stress levels in medical students but further research is required.[5] Heavy flow and family history also showed a greater risk for PD, as did attempts at weight loss presumably due to nutrional deficiencies but in this case too, more research is needed for scientific evidence.[5]

Though poorly understood, PD is considered to be due to prostaglandins (PG) produced in the uterus, suggested as early as 1965 by Pickles and associates.[8] Progesterone stabilizes lysosomes in ovulatory cycles, which decreases before menstruation, releasing phospholipase A2 from the

lysosomes. Phospholipase A2 will gives rise to the Arachidonic acid which is broken into PGs and leukotrienes.[2] The main PGs are PGF2α and PGE2. PGF2α is a strong stimulator of smooth muscle contraction,[7] causing myometrial contractions which produce ischemia leading to pain. This myometrial contraction also leads to increased nerve sensitivity. PGs also lower the pain threshold. This highlights why ovulatory cycles are associated with dysmenorrhea.[1,9]

Though there is inadequate literature to establish a single etiology, multiple factors have been found, including increased PGF2α and increased Vasopressin (VP).[2] VP is a posterior pituitary gland hormone, the level of which varies in the MC, and it may also play a role in myometrial contractility and ischemia.[2] Plasma PGF2α metabolites were found in significantly greater levels in the group with dysmenorrhea, in contrast to the non-dysmenorrheic group,[2,8] and women with greater symptoms have higher levels.[9] Plasma levels of VP and IL-6 were also reported to be higher in the dysmenorrheic group when compared with the women without dysmenorrhea.[10] VP receptor blockers have shown pain relief, but the results have not been consistent.[1,9] Higher C-Reactive Protein (CRP) levels are found during menstruation compared to other parts of the monthly cycle but the increased levels are not related to the hormonal changes in MCs,[7] and dysmenorrheic women had significantly higher levels than non-dysmenorrheic women.[11]

A study showed that 23.6% of dysmenorrheic university students avoided using any pharmacological methods to relieve their pain, of which 22.9% abstained from analgesics due to a fear of side effects such as vomiting, diarrhea, constipation, headache and drowsiness that were reported in their study.[5] In Traditional Chinese Medicine (TCM), acupressure is a technique involving pressure application on the body with a finger, thumb, or device at specific points (known as acupoints).[12] Systematic reviews studying the effects of acupressure have found support for its significant effectiveness.[12] Studies have shown that acupressure relieves pain by the production of endogenous analgesics by stimulating the autonomic nervous system.[13]

Few studies have delved into the precise events that mediate these outcomes. Specifically, there is a need for comprehensive research on how acupressure modulates neurohormonal pathways it activates, beyond general opioid peptide and endorphin release. So, the present research aimed to study the effects of acupressure on plasma levels of CRP, IL-6, PGF2α, and VP. To find changes in plasma levels, if any, in CRP, IL-6, PGF2α, and VP after applying acupressure therapy in the PD participants.

METHODOLOGY

A quasi-experimental design was used. A single group was studied, measuring the pre and post-acupressure levels of the biochemical markers mentioned above. The research was approved by the ethical committee of Baqai Medical University (ERC number BMU-EC/04-2022), and the "Code of Ethics of the World Medical Association" (Declaration of Helsinki) was taken into consideration while conducting the research. Data was collected from fifty participants of a private medical college in Karachi in a duration of eight months between September 2022 and April 2023. Participation was purely voluntary. The participants were explained in detail the requirements of the study, after which a written informed consent was taken. A modified menstrual questionnaire was distributed to select participants. It inquired about age, height, weight, marital status, menarche age, number of bleeding days, and regularity. Closed-ended questions were used to exclude diabetes, hypertension, pelvic disease or surgery, any neuropathies, and stress or depression. For menstrual pain characteristics and analgesic/alternative method use, a Likert scale was used with the options; never, occasionally, sometimes, often, and always.

Openepi was used to calculate the sample size by a professional statistician. A sample size of twenty before and twenty after intervention was obtained with a Confidence Interval (two-sided) of 95% and Power of 80%. To strengthen the study, we recruited an additional thirty students, increasing the sample size to fifty participants.

For blood testing, ELISA kits were used; Human Prostaglandin F2α ELISA kit, Cat. No. E0973Hu (BT laboratory, Birmingham, UK), Human Interleukin 6 ELISA Kit, Cat. No. E0090Hu (Li StarFish srl, Italy), Human antidiuretic hormone Kit, Cat. No. E1048Hu (Li StarFish srl, Italy) and Human C-Reactive Protein ELISA Kit, Cat. No. E1798Hu (BT laboratory, Birmingham, UK).

Convenient sampling was used. Students at a private medical institute who suffered from dysmenorrhea every single month and did not use a painkiller were recruited. See Figure 1 for the diagrammatic representation. 250 questionnaires were distributed, of which 240 were filled out and returned. 224 students suffered from PD, out of which 112 had PD every month. Of these 112 students, 66 used analgesics while 46 did not use any analgesics. 46 students fulfilled our criteria, and four analgesic users volunteered to abstain from using them for the required duration of the study, thus bringing the total sample size to 50 students.

Inclusion criteria were students who were healthy with ages between 16 and 25, having regular, normal, menstrual cycles, experiencing PD every month, and avoiding analgesics. All categories of Body Mass Index according to the World Health Organization were included.

Exclusion Criteria[5] was any pelvic disease, any history of pelvic surgery, diabetes mellitus, hypertension, a recent stressful incident, depression or mental illness, any other co-morbid conditions, any neuropathies or injury in the lower leg.

The students were taught about the self-application of

acupressure at SP6 by a certified acupressurist. SP6 acupoint is located four fingers above the medial malleolus between the tibia bone and the muscle.[14] This acupoint is shown in Figure 2. After the first blood sample (pre-intervention), the participants applied acupressure with their finger or thumb during the three subsequent MCs during PD on both legs, one by one, for two to five minutes each. The participants reported no adverse effects of acupressure.

Blood samples were collected in the first month of the study on the first day of the MC without intervention, which comprised the pre-acupressure samples. Then during the third month of acupressure application, the post-intervention blood samples were taken on the first day of MC approximately one hour after acupressure. Samples were stored at -20^{0} C till further analysis.

Data Analysis was done using SPSS version 23.0. Means with standard deviations of the baseline characteristics were calculated. The median (Interquartile Range – IQR) of the biochemical marker levels were compared before and after intervention. The Wilcoxon Sign Rank test was used instead of the paired sample t-tests as the data was not normally distributed due to some outliers.

RESULTS

Table 1 reports the baseline characteristics of studied samples, in the present study there were 50 participants, Mean Age (years) was 20.92 (SD=±1.56), mean Weight (kg) was 54.6 (SD=±10.8), mean Height (m) was 1.615 (SD=±0.063), mean BMI (kg/m2) was 20.91 (SD=±3.6), mean Age of Menarche was 13.28 (SD=±1.58), mean Number of bleeding days was 5.30 (SD=±1.38).

Table 2 reports the pre and post-acupressure plasma levels in the PD group. The Wilcoxon Signed Rank test was used in place of paired sample t-tests because the data were not normally distributed due to some outliers. The pre-acupressure PGF2α Median was 383.4(IQR=98.9), IL-6 Median was 75.49(IQR=35.87), VP Median was 50.07(IQR=24.48), and CRP Median was 0.66(IQR=0.4019) and post-acupressure PGF2α Median was 374.3(IQR=360.2), IL-6 Median was 72.18(IQR=23.71), VP Median was 50.32(IQR=26.09), and CRP Median was 0.8(IQR=0.4858). The Wilcoxon signed-rank test showed a significant increase in CRP after treatment ($p = 0.03$), while no significant changes were found in PGF2α, IL-6, and VP markers ($p > 0.05$).

DISCUSSION

Acupressure is a type of massage, and massage has been shown to lower the production of IL-6 along with other products. Thus, acupressure may decrease the inflammatory responses, both local and systemic.[15] There is evidence that acupoint stimulation produces a variety of effects, including changes in neurohormone secretion.[16] Studies regarding the effects of acupressure on plasma levels of biochemical markers in PD were not found. On the basis of the available

Figure 1. Diagrammatic description of the recruitment and induction of study subjects

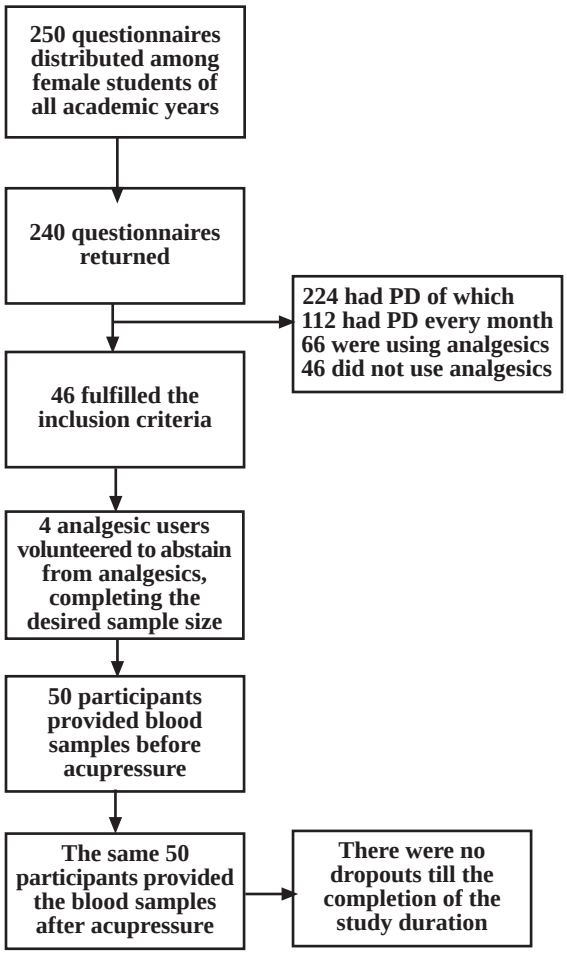

Figure 2. Location of the acupoint Sanyinjiao Point 6 (SP6). (Photograph taken of a study participant with consent).

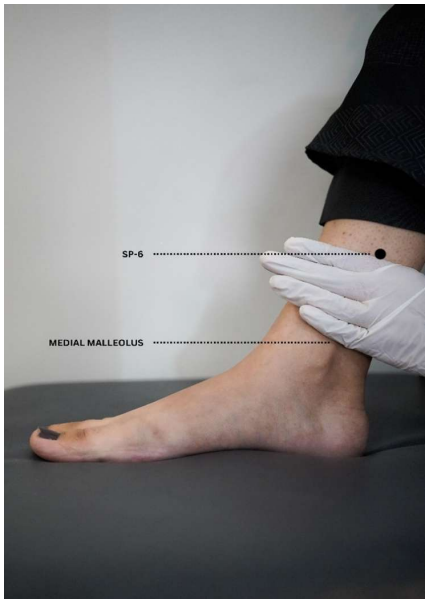

Table 1: Baseline Characteristics of Studied Samples (n=50)

PARAMETERS	*Mean±SD (N=50)*
Age (years)	20.92±1.56
Height (m)	1.615±0.063
Weight (kg)	54.6±10.8
BMI (kg/m^2)	20.91±3.60
Duration of bleeding (days)	5.30±1.38
Age of Menarche (Years)	13.28±1.52

Table 2: Comparison of pre and post-acupressure levels of PGF2a, IL-6, VP, and CRP

Parameters	*Median (IQR) Pre-acu*	*Median (IQR) Post-acu*	*P-value*
PGF2a pg/ml	383.4 (464.3-365.4)	374.3 (698.8-338.6)	0.60
IL-6 pg/ml	75.49 (103.9-68.03)	72.18 (88.52-64.81)	0.84
VP pg/ml	50.07 (68.43-43.95)	50.32 (70.59-44.5)	0.19
CRP mg/L	0.66 (0.8976-0.4957)	0.80 (1.061-0.5752)	0.03*

*P<0.05 was considered statistically significant using the Wilcoxon Signed Ranks test

IQR: Interquartile Range, Pre-acu: Pre-acupressure, Post-acu: Post-acupressure, PGF2á: Prostaglandin F2á, IL-6: Interleukin 6,VP: Vasopressin, CRP: C-Reactive Protein

literature and with the best of our knowledge, we can say that this is the first study to determine the influence of acupressure at SP6 on plasma levels of PGF2α, VP, CRP, and IL-6 in women with PD.

In this study, despite significant pain relief,[17] only CRP showed a statistically significant greater value in the post-acupressure plasma levels (p=0.03). Interestingly, CRP is known to be an inflammatory marker and is higher during menstruation as pointed out earlier, but its increased level after acupressure despite pain relief appears contradictory. On the other hand, IL-6, also a known inflammatory marker, showed no increase. This may mean that the CRP rise was not necessarily inflammatory-mediated, or it rose via a non-IL-6 pathway. There may be confounding factors other than those excluded (coexisting morbidity, infection, injury, and known chronic stress) during the selection of the participants. The other confounding factors may be physical exertion, temporary stress, sleep disturbance, subclinical undiagnosed infection or diet-based factors. Laboratory or handling errors may also partially contribute. The fact that there was significant pain relief suggests that the pain relief is not due to decreased inflammation (as endometrial sloughing persists), it is more likely neuromodulated, but further research is needed with larger sample sizes. Other studies observing the effects of acupressure on CRP were not found, albeit a Polish study studied the effects of manual therapy on PD[7]. In contrast to the present study, they found no difference in CRP plasma levels after manual therapy in PD despite pain relief. Their sample size was 20 with similar baseline characteristics.

In the current study, PGF2α plasma levels showed a slight decrease in the post-acupressure levels but were not statistically significant (p=0.60) despite significant pain relief. This may lead to the speculation that the pain reduction was not PGF2α mediated, even though PGs, especially PGF2α sensitize nerve endings to pain.[18] No studies were found where PGF2α was measured pre and post-acupressure in PD. One study in Poland[7] compared PGF2α plasma levels in PD before and after "manual therapy" and reported no significant difference despite significant pain relief similar to our findings. Available data suggests that the pain relief by manual therapy is due to the vagal nerve stimulation. Through the stimulation of the parasympathetic nervous system, there is vasodilation in the pelvis which relieves the ischemia.[7]

Conversely, an Indonesian study reported PGF2α changes in PD after Slow Stroke Back Massage (SSBM), Warm compress, and Cold compress, but NOT acupressure.[19] Significantly decreased levels of PGF2α levels were found in the intervention group (p=0.01) when compared with the control group. (n=13 per group). SSBM is a massage over the whole back, while acupressure is a type of massage only at specific points. Likewise, temperature changes occur with warm and cold compresses that may affect the synthesis and metabolism of PGF2α.

Also in opposition to our findings, Kannan and co-workers found a reduction in metabolites of PGF2α and pain after aerobic exercise in PD and concluded that the pain relief may be chemically mediated.[20] The age range of the participants was similar to the current study but their sample size was 10 for the exercising group and 10 for the control group.

Tyas and his coworkers compared PGF2α levels in a younger reproductive age group (11-17 years) before and after abdominal stretching, dysmenorrhea exercises, and yoga.[21] They reported a significant reduction after dysmenorrhea exercises and yoga, but significant pain reduction was reported only after the dysmenorrhea exercises. Thus implying that pain relief is not solely dependent on chemical mediation supporting our finding. Another research regarding acupuncture with auricular acupressure effects on menstrual headaches, NOT PD, found significant reductions (p<0.01) in plasma PGF2α levels.[22]

The present study found no significant difference between the VP levels before and after intervention (p=0.19). Other studies measuring VP before and after acupressure for PD were not found, based on the current knowledge we have. One study showed that acupuncture with auricular

acupressure reduced the VP levels, but this research studied the effects on menstrual headache, NOT PD.[22]

In the current study, post-acupressure IL-6 levels revealed a mild reduction but not significant (p=0.84). Other studies where IL-6 levels after acupressure were evaluated in PD were not found. Rahimi and coworkers compared pain and blood levels of IL-6 before and after 2 months of high-intensity interval and moderate continuous training on PD. The sample size of the study was 30 and had a similar age range to our study. They found no significant difference in the IL-6 levels.[23] In contrast, a study regarding acupressure effects on low back pain, not PD, found a rise in IL-6 levels after auricular acupressure when compared with a control group.[24] Their sample size was 19, of which 10 used auricular acupressure and 9 used a sham acupressure and the age of participants was over 40 years as they studied low back pain and not PD. A study by Yeh in 2023 supports the role of inflammatory markers in analgesia by acupressure.[25] They correlated auricular point acupressure, pain intensity (chronic low back pain) and inflammatory markers that included IL-2, NOT IL-6. A significant decrease was found in the pre and post-acupressure levels of IL-2 as opposed to our results.

There is scanty literature available on the current topic, so there is a crucial need to evaluate the mechanism of analgesia mediated by various hormones, cytokines, and other inflammatory markers in PD to enhance the level of comfort among a diverse population of women. Non-significant results are not the limitations of the study, because it pointed out an important cost-effective, reliable, authentic, and independent method for pain suppression mediated by specific biomarkers. However, an innovative mechanism can be more clearly identified by the implication of a more generalized study design.

Strength of the study: The strength of the study is, from our perspective, that it is probably the first study of this specific type in Pakistan and the South Asian region.

Limitations: The limitation of this study is that the participants included all medical students, so we can not generalize the findings to the total population.

CONCLUSÝON

The study findings suggest that the amelioration of pain by acupressure at SP6 in PD is not purely mediated via biochemical markers, and other pathways are also involved. Continued research is required to comprehend the complexity of the association of biomarkers with analgesia via acupressure. Future recommendations are to conduct studies to map the precise cellular pathways through which PGF2α vasopressin, IL-6, and CRP contribute to uterine contractility and pain. This could involve studying signalling cascades in myometrial and endometrial cells. Investigate how signalling might interact at a molecular level, such as receptor co-activation or shared downstream targets in uterine tissue.

Authors Contribution:
Samira Amjad: Conceptualized the study, planned the design, methodology, and data curation, wrote the manuscript and statistical analysis, and is responsible for the integrity of the research.
Qamer Aziz: study design, and assisted in the result interpretation, critically reviewing, and approval of the final version of the draft.
Ruqaya Nangrejo: study design, data analysis, and drafting the article

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