The Effects of Cold Compress and Warm Compress on β-Endorphin Levels, IL-6 and TNFα among Adolescent with Dysmenorrhea

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The Effects of Cold Compress and Warm Compress on β-Endorphin Levels, IL-6 and TNFα among Adolescent with Dysmenorrhea

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ABSTRACT

Non-pharmacological efforts to treat dysmenorrhoea are include cold compresses and warm compresses. The aim of this study was to determine the differences effect of cold compresses and warm compresses to β-Endorphin levels, IL-6 and TNFα among adolescents with dysmenorrhoea. The research was Post Test Only with Control Group. β endorphin, IL-6 and TNFα were measured by ELISA, then analyzed by Independent Sample T-Test. The average β level of Endorphin in cold compress group was 143.03 pg/ml, in warm compress group was 171.43 pg/ml; the average IL6 level in cold compress group was 1352.60 pg/ml, in warm compress group was 961.14 pg/ml and the average TNFα level in cold compress group was 345.75 pg/ml, in warm compress group was 262.50 pg/ml. The results of Independent Sample T-Test showed that there was no difference in β levels of Endorphin IL-6 and TNFα in both of the warm and cold compresses group. Cold compress and warm compress can stimulate loose of Endorphin β levels and regulate uterine hypercontractility during menstrual pain. Cold compress and warm compress can be used as an alternative to treat dysmenorrhoea.

Keywords: *Cold compress, Warm compress, β-Endorphin levels, IL-6 levels, TNFα levels*

INTRODUCTION

Dysmenorrhoea is a painful sensation with cramps sensation in the lower abdomen and commonly followed by sweating, tachycardia, headache, nausea, vomiting, diarrhea, and back pain before or during menstruation[1-3]. The intensity of menstrual pain was varies from mild, moderate and severe[4]. Severe of dysmenorrhea give affects physical, psychological and social consequences[5].

The prevalence of dysmenorrhoea in the world was varies from 37% to 90.1%, in China there were 37%[6], 55.5%-70% in adolescents and young adults in Turkey[7-8], 60.9% of female medical students in King Abdulaziz University[9], 74.4% in teenage girls in Ghana[10], 74%-86.1% in Iran, 77.6% among University of Gondar Students, Northwestern Ethiopia[11], 90.1% among Jordanian University students[12]. In Indonesia an estimated 55% of women in productive age were experienced menstrual pain[13]. In East Java, the number of reproductive young women aged 10-24 is 56,598 and about 11565 (1.31%) of those experienced dysmenorrhea and come to the obstetrics[14].

Factors that can increase the risk of dysmenorrhoea are include age and age of younger menarche, longer duration of menstruation, menstrual volume[15-17], low of BMI, smoking and alcoholism[16,18-19], low social support, family history of dysmenorrhoea, high caffeine consumsion[20], depression, anxiety and stress[7,21]. Primary dysmenorrhoea has a biochemical basis and doe to prostaglandin loose during menstruation. During the luteal and menstrual phases, prostaglandin F2-alpha (PGF2-α) were excretion. Excessive release of PGF2-α will increase the amplitude and frequency of uterine contractions and causes vasospasm of the uterine arterioles, causing lower abdominal ischemia

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and cramps and back pain[23]. Psychiatric factors also play a role in the occurrence of primary dysmenorrhea. Stress can increase the levels of vasopressin and catecholamines and it will make vasoconstriction and ischemia in cells[24]. Peripheral blood analysis in women with dysmenorrhoea shows excessive synthesis and concentration of oxytocin, PGF2-a, vasopressin, IL-6 and TNF[25-28]. Dysmenorrhoea is a major cause of activities problem[29] such as absent from work or school[30-31] and decreased quality of life[8,32-33].

Pharmacological interventions for dysmenorrhoea use nonsteroidal anti-inflammatory drugs (NSAIDs) and oral contraceptive. The side effects including dependence[34-35], diarrhea, abdominal pain, nausea[36], kidney and liver complications, sleep disorders[37], digestive disorders[38]. The failure rate of pharmacological treatment is 20-25%[39]. Non-pharmacological interventions include cold and warm compress. Cold compress is ice therapy that can reduce prostaglandins which strengthens pain sensation and other subcutaneous at the injury place by inhibiting the inflammatory process. This is because cold compress can reduce blood flow to a part and reduce bleeding edema which is it cause analgesic effects by slowing the speed of nerve delivery so the pain impulses will less reach to the brain[40]. Warm compresses with hot jars cause conduction, where there is transfer of heat from the bladder into the body and it giving dilation for blood vessels and decreased muscle tension so that dysmenorrhoea pain will be reduced[41]. Skin stimulation causes the release of endorphins, thus blocking the transmission of pain stimuli[41]. The results of previous studies showed that Moxibustion can reduce the levels of PGF2α, oxytocin, vWF and increasing the levels of β-EP. The effect of cold and warm compress on β-Endorphin, IL-6 and TNFα has not been clearly known, so the researchers are interested to conducting the research about The Effects of Cold Compress and Warm Compress on β- Endorphin levels, IL-6 and TNFα among Adolescents with Dysmenorrhoea.

MATERIALS AND METHOD

The design of this research was Pretest-Postest. The population were all students at FIK-Unipdu Jombang who experienced dysmenorrhoea. Sample size was 40, selected by purposive sampling, then divided into cold compress group (n=20) and warm compress group (n=20). The instrument of data collection were thermometer, a hot jar and ice bag. Numeric Rating Scale used to measure pain level. ELISA inderect method to measure the levels of β Endorphin, IL-6 and TNFα using the. Data were analyzed by T-Test.

FINDINGS

The intensity of dysmenorrhoea before giving cold compress were mostly at moderate. However, in warm compresses group were more than half of participant at severe level. Intensity of dysmenorrhoea after giving treatment in cold compresses group were mostly at mild, while in warm compresses group were mostly at moderate level. Homogeneity of variances test results showed that the intensity of dysmenorrhoea before and after giving treatment in both of groups were not have a significant difference.

Table 1. The differences of β-Endorphin levels, IL-6 and TNFα after giving intervention

Variable	Cold compress Mean-(SD)	Warm compress Mean-(SD)	Mean Difference (95%-CI)	p
β-Endorphin	143.03(3.97)	171.43(2.59)	-28.40(-59.88=3.08)	0.074
IL-6	1352.60(3.57)	961.14(3.79)	39.46(-38.15-821.01)	0.070
TNFα	345.75(1.55)	262.50(6.14)	83.25(-42.85-209.35)	0.179

There were no have significant differences levels of β Endorphin, IL-6 and TNFα after giving treatment

β-endorphin levels after giving cold and warm compress had no significant differences. Cold and warm compress are the techniques for cutaneous stimulation. Cutaneous stimulation is skin stimulation carried out to relieve pain, works by encouraging the release of endorphins, so it will block the transmission of pain stimuli[41]. Changes in β-Endorphin levels can be explain the basis of Opiate Endogenous theory, where opiate receptors in the brain and spinal cord were determine the central nervous system to activate morphine substances called endorphins and enkephalin when pain is received. This endogenous opiate can be stimulated by skin stimulation and muscles. These opioid receptors are located on peripheral sensory nerve extremity[42].

Cold compress was given by using an ice bag filled with ice, compressed to the abdominal area for minutes and a warm compress was given by using a bag filled with warm water at a temperature of 40-45 C° and compressed to the abdominal area for 20 minutes. The average β-endorphin level at cold compress group was 143.03 pg/ml, the warm compress group was 171.43 pg/ml. Giving cold and warm compresses can increase β-endorphin levels to relieve pain production. The higher of endorphins level make the level of pain at mild [43]. Endorphins inhibit fiber C in pre and post synapses and Aδ fibers in the dorsal horn and activate the larger of Aß (A-beta) sensory nerve fibers, thus blocking the pain signals when enter to spinal cord so the pain perception will decreases[44]. After intervention, the intensity of dysmenorrhea among respondents will decreased. This because of the release of β-endorphins levels that inhibit C fiber and activate Aß sensory nerve fibers so it will inhibits the pain signals to spinal cord and decreased perception of pain. The result was in accordance with previous studies which showed that β-endorphin levels in primary dysmenorrhoea increased after moxibustion therapy. Moxibustion therapy is a warm moxa stimulation at Guanyuan, Shenque and Sanyinjiao acupuncture points, the treatment giving for 10-15 minutes a day during 7 days before menstruation in 3 menstrual cycles[45].

IL-6 and TNFα levels had no difference. In primary dysmenorrhea, the level of genes expression of cytokine pro-inflammatory (IL1B, TNF, IL6 and IL8) at the first day of menstruation will significantly increases[28]. IL-6 functions to increase oxytocin secretion at the first day of menstruation[46], where TNFα functions to increase prostaglandin and oxytocin at the first day of menstruation[47-48]. Increased prostaglandins and oxytocin have an impact to excessive uterine contractions, decrease endometrial blood flow and cause pain during menstruation[28].

Cold compresses provide physiological effects to reduce the inflammatory response, blood flow and edema, local pain[49]. Heat will stimulates the vascular reaction by increasing blood flow, resulting in delusions prostaglandins, bradykinin and histamine. Increasing blood flow also can increase oxygenation[39]. Local heat will give the abdomen to increasing gastrointestinal motility and relaxation to the uterus. Local heat is as effective as NSAIDs[50]. NSAIDs can reduce the accumulation of prostaglandins and reduce spasmodic contractions caused by prostaglandins and inhibit the activity of COX-2 and COX-1 enzymes[51].

The results of previous studies showed that the giving of warm stimuli (moxibution) can regulate uterine hypercontractility during menstrual pain by set of the mediator pain level serum where occur the decreasing levels of PGF2α serum and oxytocin[45]. The effect of moxibution treatment works like electroakupunctur[52]. Several studies have shown that electroacupuncture can reduce the expression of prostaglandin levels[53], peripheral blood lymphocytes among rat as the samples with primary dysmenorrhoea[54]. T-cells are the main source of cytokine secretion (TNF, Interleukin, interferons)[55]. Thus the cold compresses and warm compresses interventions can reduce pro-inflammatory cytokines IL-6 and TNFα.

CONCLUSION AND RECOMMENDATION

The results of this study showed that after giving warm and cold compresses in both group there were no differences in levels of β Endorphin, IL-6 and TNFα among adolescents with dysmenorrhoea. Cold compresses and warm compresses can be used as an alternative treatment to dysmenorrhoea.

Ethical Clearance: Ethics Committee of Nursing Faculty, Airlangga University

Conflict of Interest: No

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