Letter to Editor

Role of Cold Application in Primary Dysmenorrhea

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Dear Editor,

We read Ajmi et al.'s article with great interest. The authors discussed the temperature-dependent effects of hip baths on primary dysmenorrhea patients through a randomized controlled study [1]. The study aims to investigate the impact of specific temperatures of hip baths on individuals with primary dysmenorrhea. Participants were randomly divided into three groups of 60: Cold Hip Bath (CHB, 10 to 18°C), Hot Hip Bath (HHB, 40 to 45°C), and Neutral Hip Bath (NHB, 32 to 36°C). Each intervention was administered for 20 minutes per session, over three months, with a cold compress on the head. The interventions began on the sixth day of the menstrual cycle and continued until the following cycle. The CHB group showed a significant reduction in pain, with Visual Analog scale (VAS) scores dropping from 7.27 to 2.07 and Menstrual Symptom Questionnaire (MSQ) scores falling from 72.87 to 64.20, along with improvements in haemoglobin, red blood cell counts, and reductions in eosinophils and lymphocytes. The HHB group also showed significant pain reduction (VAS: 7.05 to 4.15, MSQ: 73.12 to 65.27) and improvements in blood parameters, including increased haemoglobin, platelets, and Mean Corpuscular Volume (MCV). The NHB group reported moderate pain relief (VAS: 7.28 to 5.95, MSQ: 72.48 to 69.20) with minimal eosinophil changes. According to comparative analysis, cold hip baths are more effective than hot or neutral baths in reducing pain intensity and menstrual symptoms, as measured by the VAS and MSQ scales.

Hydrotherapy uses water in various forms with varying temperatures to produce different physiological effect. Cold water interventions were used across disciplines to improve physiological and psychological well-being.

Although the authors effectively explained several mechanisms of pain reduction in patients with primary dysmenorrhea, we would like to highlight the role of cold application in reducing prostaglandins, which are major cause of dysmenorrhea. Primary dysmenorrhea is the most painful and severe condition that affects women during menstruation. Although the exact pathophysiology of primary dysmenorrhea is unknown, several studies have evidence regarding prostaglandin's role in primary dysmenorrhea. Prostaglandins are synthesized from arachidonic acid, which is released from membrane phospholipids during the menstrual cycle. This process is primarily mediated by the enzyme cyclooxygenase (COX). Hormonal changes, particularly progesterone, had a significant influence on prostaglandin production. During the luteal phase of the menstrual cycle, progesterone levels peak. If fertilization does not occur, progesterone levels decline sharply, leading to endometrial sloughing.

This process causes lysosomal enzymes to produce arachidonic acid, which increases prostaglandin synthesis. Prostaglandin F2α (PGF2α) and Prostaglandin E2 (PGE2) are the key prostaglandins involved in primary dysmenorrhea.. Both of these promote myometrial contractions necessary for expelling the uterine lining. However, when prostaglandin levels are high, contractions become stronger and more painful, resulting in ischemia (reduced blood flow) and pain. Furthermore, the increased prostaglandin levels stimulate nerve endings in the uterus, increasing pain perception. This hypersensitivity often leads to significant discomfort that peaks during the first days of menstruation, when prostaglandin levels are at their highest [2]. Research indicates that women suffering from primary dysmenorrhea exhibit higher concentrations of both PGF2α and PGE2 in menstrual fluid compared to those without dysmenorrhea [3].

The intervention focuses on lowering prostaglandin levels, which will play important role in reducing menstrual symptoms and pain. Despite the authors success in explaining several mechanisms for pain reduction in primary

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dysmenorrhea patients, the role of prostaglandins remained unclear. So, we would like to suggest an additional mechanism in which the prostaglandins play major role in primary dysmenorrhea patients receiving cold hip baths. The positive effects of cold application on dysmenorrhoea may be due to one of the following mechanisms:

(i) Cold exposure induces a metabolic shift from primarily lipid metabolism to increased carbohydrate utilization. This is particularly evident in brown adipose tissue (BAT), which activates during cold exposure to generate heat through non-shivering thermogenesis. This process increases glucose uptake and improves insulin sensitivity, which may reduce overall metabolic activity [4]. The decrease in metabolic activity during cold exposure leads to reduced synthesis of free fatty acids (FFA) and triglycerides. Prostaglandins, which are synthesized from arachidonic acid derived from membrane phospholipids, are frequently secreted response to tissue injury and inflammation [5]. A reduction in lipid availability can therefore lead to decreased prostaglandin synthesis, which in turn reduces primary dysmenorrhea symptoms**.**

(ii) Cold exposure activates the sympathetic nervous system, which results in vasoconstriction followed by vasodilation. This process involves complex neurogenic mechanisms in which sympathetic nerves release neurotransmitters that regulate vascular tone. The subsequent vasodilation allows for increased blood perfusion to tissues that would otherwise be without adequate blood supply during cold exposure [6]. In addition to neural mechanisms, local factors such as nitric oxide (NO) promote vasodilation during rewarming phases after cold exposure. Increased NO production improves endothelial function and contributes to vascular relaxation, promoting increased blood flow and clearance of inflammatory substances such as prostaglandins [7]. Cold hip baths can clear prostaglandins from the tissues by increasing blood flow to the hip and pelvic region. Reduced prostaglandins alleviate the menstrual symptoms and pain thresholds associated with during primary dysmenorrhea [8].

(iii) While acute cold exposure may initially increase PGE2 levels as part of the thermogenic process, Prolonged cold exposure can alter hormone levels, including increased secretion of fibroblast growth factor 21 (FGF21). FGF21 enhances metabolic efficiency and may inhibit inflammatory pathways, such as those involving COX and PGE2 synthesis [9]. When exposed to prolonged low temperatures, the body may develop feedback mechanisms that reduce COX activity. This could involve changes in gene expression related to COX enzymes or alterations in substrate availability for PGE2 synthesis [10]. Reducing PGE2 levels may reduce symptoms associated with primary dysmenorrhea.

Cold hip baths (10–18º C) for 20 minutes would reduce the prostaglandin levels. The mechanisms described above for prostaglandins may strongly support the use of cold hip baths to treat primary dysmenorrhea. We recommend cold hip baths as an adjuvant treatment because they are a simple and cost-effective intervention compared to other methods of treatments. Educating women of menstrual age about this simple approach may significantly improve their quality of life by effectively modulating prostaglandin levels and reducing associated menstrual pain.

In conclusion, the positive outcomes demonstrated in the study by Ajmi et al. strongly support the use of cold hip baths as an alternative treatment for primary dysmenorrhea. CHB intervention aligns with a growing emphasis on non-pharmacological approaches to primary dysmenorrhea management.

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