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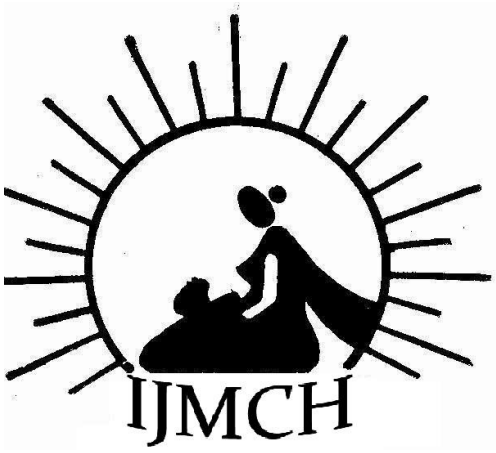

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Fertility hormones regulate synthesis of prostaglandins responsible for uterine muscle contraction causing pain during menstruation.

Prevalence of hyperprolactinemia in Primary Dysmenorrhoea

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Abstract

Background: Fertility hormones regulate synthesis of prostaglandins responsible for uterine muscle contraction causing pain during menstruation. Dysmenorrhoea apart from being distressing may be having associated pathologies and hormone imbalance. So the patients were examined for galactorrhoea, prolactin (PRL) and thyroid stimulating hormone (TSH) levels.

Materials and Methods: 100 patients of primary dysmenorrhoea visiting OPD of Obstetric & Gynecology Department of tertiary care hospital were enrolled for the study (group A). 100 age matched females without dysmenorrhoea and any gynae complaints were taken as controls (group B). Detailed history of dysmenorrhoea, menstrual cycle and breast discharge was taken. All the patients were assessed for pain on VAS scale and effect on daily activity and analgesic requirement was noted. TSH and PRL levels of all the subjects were analyzed. Sonography of pelvis was done to rule out any pathology.

Results: The mean age of presentation was 21.58 ±4.68 years. Study group patients had significantly higher incidence of galactorrhoea (30% vs 10%) and hyperprolactinemia(20% vs 5%) compared to control group. Incidence of hypothyroidism was also higher(10% vs 3%).
Conclusions: Significant number of dysmenorrhoea patients presented with breast discharge and high levels of TSH and PRL.

Recommendation: All dysmenorrhoic patients should be examined for galactorrhoea, hyperprolactinemia and subclinical hypothyroidism, as the hormonal changes might affect fertility.
Key words: Dysmenorrhoea, galactorrhoea, hyperprolactinemia, subclinical hypothyroidism, prolactin (PRL), TSH.

Introduction

Dysmenorrhoea is severe painful frequent cramping during menstruation. It has impact on personal life and productivity, hence is distressing. It usually appears within 1-2 years of menarche, when ovulatory cycles are established. These patients have higher levels of prostaglandins, (PGF2α and PGE2) which causes increased contractions of uterus responsible for severe pain and heavy bleeding.[1-2] Dysmenorrhoea can be without an organic cause (primary dysmenorrhoea) or with organic pathology (secondary dysmenorrhoea). Primary dysmenorrhoea is more prevalent than secondary dysmenorrhoea.[3] It may be related to hormonal changes since it occurs in ovulating females. Its association with hormonal changes has been poorly studied. A study showed correlation of hyperprolactinemia with primary dysmenorrhoea.[4] Hyperprolactinemia cause galactorrhoea and menstrual disorders like amenorrhea and infertility. Hyperprolactinemia and galactorrhoea leads to estrogen deficiency due to inhibition of gonadotropin releasing hormone and subsequent inhibition of luteinizing and follicular stimulating hormone.[5] These may cause insufficient progesterone secretion leading to luteal phase deficiency, infertility and prostaglandin imbalance causing dysmenorrhoea. There is an association of hyperprolactinemia, galactorrhoea and hypothyroidism, these patients should be monitored for TSH levels as well.[6] So the present study was undertaken to evaluate presence of galactorrhoea and role of hormones PRL and TSH in dysmenorrhoea.

Materials and Methods

100 patients of primary dysmenorrhoea (15-30 years) visiting OPD of Obs & Gyne Department of tertiary care hospital were enrolled for the study (group A). 100 age matched females without dysmenorrhoea and any gynae complaints were taken as controls (group B). A detailed history of dysmenorrhoea (time of onset, subsidence, duration), menstrual cycle and breast discharge was taken. Sleep disturbance and urinary or bowel complaints were also recorded. All the patients were assessed for pain of dysmenorrhoea on VAS scale and its effect on daily activity and analgesic requirement was noted. A detailed systemic and bi manual examination was done.

Fasting serum TSH and PRL levels were analyzed by chemiluminescence on Elecsys. 2010 (Roche). Sonography of pelvis was done to rule out any pathology. Patients with any organic pathology were not included in the study.

Statistical Analysis

Mean and standard deviation were computed. The difference between two groups was seen by applying t-test. The level of significance considered was 0.05.

Results and Discussion

Primary dysmenorrhoea occurs without an organic pelvic pathology. It can be severe and incapacitating. These patients have higher levels of prostaglandins, (PGF2alfa and PGE2) which causes increased contractibility of uterus leading to dysmenorrhoea.[3] These compounds are formed in higher concentration in secretory endometrium than in proliferative endometrium. The decline in progesterone levels in late luteal phase triggers lytic enzymatic action resulting in release of phospholipids with generation of arachidonic acid and activation of cyclo oxygenase pathway leading to dysmenorrhoea.[7]

Dysmenorrhoea may begin soon after menarche and often improves with age, or it may originate later in life due to onset of underlying causative conditions.[8] This is consistent with our study as the mean age of presentation was 21.58±4.68 years. Majority of the patients were unmarried in our study. Severity of dysmenorrhoea decreases with age and childbirth [9] which correlates with our study, as only 10% patients reported dysmenorrhoea after child birth Table 1.

Incidence of hyperprolactinemia (20% vs 5%) and galactorrhoea (30% vs 10%) was significantly higher in study group as compared to control group ($P<0.05$ and $p<0.001$ respectively). Association of galactorrhoea with dysmenorrhoea has not been reported earlier Table 2. Dysmenorrhoic women have shown to be having higher levels of estrogen leading to stimulation of PRL secretion .[10] High PRL levels as noted in our study (levels ranging from 25-40 ng/dl), could be due to physiological causes like stress, suckling, exercise, sexual activity leading to galactorrhoea and premenstrual tension.[5] So hyperprolactinemia could be cause of galactorrhoea as reported by some workers.[11] But all patients with galactorrhoea did not have high prolactin levels in our study, as clinical hyperprolactinemia may show normal PRL levels because of heterogeneity of the peptide hormone (immunoactive and bioactive forms).[12] It has well characterized effects on the normal gonadal function and impair fertility at different levels.[13] 10% patients had hypothyroidism in study group and in control group only 3% patients had high TSH levels Table 2. Thyroid disorders can affect fertility in various ways resulting in anovulatory cycles, luteal phase defect, high PRL levels and sex hormone imbalances.[14] Thus there is a need of evaluating patients for galactorrhoea, PRL and TSH levels as to prevent future infertility and osteoporosis.

Conclusions

The prevalence of dysmenorrhoea has been reported very high(50%). Age of presentation is 21.58±4.68 years, fertility can be an important concern at this age (marriageable age). It is recommended that all patients with dysmenorrhoea must be examined for galactorrhoea, sub clinical hyper prolactinemia and hypothyroidism. Hormone imbalance may be diagnosed and treated before complications of infertility begin.

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Table 1 Mean age and Gynae History of both the groups

Parameter	Group A (n=100)	Group B (n=100)
Age (years)	21.58±4.68	23.50±6.50
Unmarried	88%	75%
Married	12%	25%
Having children	10%	15%
Duration of dysmenorrhoea (days)	2.70±1.80	--
Cycle length (days)	31.94±2.54	30.56±2.84

Table 2 Percentage of patients with galactorrhoea and high PRL & TSH levels

Parameter	Group A (n=100)	Group B (n=100)
Prolactin (>25 ng/ml)	20%**	5%
TSH (>4.2µIU/ml)	10%	3%
Galactorrhoea	30%***	10%

P<0.05 -----**, p<0.001-----***.