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Contents

Leading Article

Secondary Amenorrhoea Related to Neuro-Endocrinological Causes

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Abstract

Objective: To review the neuro-endocrinological causes of secondary amenorrhoea, focusing on its pathophysiology, mediators, and management strategies.

Design: A comprehensive review of current evidence on physiological, pathological, and iatrogenic causes of secondary amenorrhoea, emphasizing functional hypothalamic amenorrhoea (FHA), hyperprolactinemia, and pituitary-related disorders.

Method: The review integrates findings from experimental studies, clinical observations, and expert consensus to outline the mechanisms underlying hypothalamic-pituitary-ovarian (HPO) axis dysregulation. Key mediators, including leptin, insulin, and fibroblast growth factor-21 (FGF-21), are explored, alongside conditions such as Sheehan's syndrome, sellar lesions, and genetic mutations.

Results: Secondary amenorrhoea affects 3–4% of women globally, with over half of cases linked to HPO axis dysfunction. FHA emerges as a leading cause, driven by stressors such as malnutrition and chronic illness. Hyperprolactinemia, both physiological and pathological, significantly impacts gonadotropin secretion and ovarian function. Pathological conditions, including pituitary adenomas, hypothyroidism, and infiltrative diseases, further contribute to amenorrhoea. Iatrogenic factors, such as cranial irradiation and medication use, also play a role. Early diagnosis and targeted interventions, including hormone replacement therapy and addressing reversible causes, are crucial for restoring gonadotropin function and improving fertility outcomes.

Conclusion: Secondary amenorrhoea is a multifaceted condition often resulting from neuro-endocrinological disturbances. A multidisciplinary approach is essential to accurately diagnose and manage the condition, ensuring optimal reproductive and overall health outcomes.

Introduction

Amenorrhoea refers to the absence of menstrual bleeding. It is a normal physiological state in pre-pubertal girls, pregnant women, those exclusively breastfeeding, and postmenopausal women. In women of reproductive age, the most common cause of amenorrhoea is undiagnosed pregnancy. When pregnancy is excluded, diagnosing the underlying cause can be challenging.[12]

Secondary amenorrhoea is clinically significant when it meets the following criteria[3]:

- Normal pubertal milestones are present.
- Menarche has been attained.
- Absence of menstruation for at least three or six months in irregular cycles.

Globally, secondary amenorrhoea affects approximately 3–4% of women.[4] Over 50% of cases are associated with dysfunction or dysregulation of the hypothalamic-pituitary-ovarian (HPO) axis.[5] This article focuses on neuro-endocrinological causes of secondary amenorrhoea, many of which are associated with coexisting fertility issues. For clarity, the etiological causes are categorized as outlined in Table 1.

Table-1; Neuro-endocrinological causes of secondary amenorrhoea

Physiological	**Pathological**	**Iatrogenic**
Functional hypothalamic amenorrhoea	Hyperprolactinemia	Pituitary surgery
Hyperprolactinemia due to pregnancy/lactation	Sellar mass/lesions	Cranial irradiation
	Sheehan's syndrome	Medication induced
	Infective or infiltrative diseases	*a. GnRH Suppression*
	Brain trauma	*b. Hyperprolactinemia*
	Adrenal pathology	*c. Hypophysitis*
	Genetic causes	

Table-2; Mediators for functional hypothalamic amenorrhoea

Leptin	A mediator of functional amenorrhoea. Secreted by adipocytes and low in FHA. Low levels associated with reduced GnRH and anovulation via Kisspeptin mediated pathway. Adequate fat stores are essential for Leptin production and is probably the basis when extreme fat loss related to FHA[10].
Insulin	Insulin has been shown to modulate the HPO axis. In obese, excess insulin with resistance creates functional hypoinsulinemia. Whereas in very thin individuals (under nutrition), there is actual hypoinsulinemia. When insulin and its receptor signaling is not properly regulated, LH level reduces and anovulation occurs[11].
FGF-21	Role in humans is unknown. But this liver derived factor is up regulated in starvation and associated with low LH and anovulation via interruption to Kisspeptin, a potent stimulator of GnRH secretion[12].

(A) Physiological Secondary Amenorrhoea

(1) Functional Hypothalamic Amenorrhoea (FHA)

Functional hypothalamic amenorrhoea (FHA), also known as stress-induced amenorrhoea, is one of the most common causes of secondary amenorrhoea. It occurs in conditions such as severe malnutrition, emotional stress, extreme physical exercise, and chronic illness. In response to these stressors, the body prioritizes essential survival mechanisms over reproductive functions, redirecting energy and resources away from the reproductive system.

The hypothalamic-pituitary-adrenal (HPA) axis becomes inactivated during periods of stress, suppressing the HPO axis. Corticotropin-releasing hormone (CRH), produced by the hypothalamus, plays a dominant role by inhibiting gonadotropin-releasing hormone (GnRH) secretion. This results in reduced pituitary secretion of follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Experimental studies have demonstrated that this effect can be reversed by external administration of GnRH, highlighting its therapeutic potential in such cases.

Additionally, elevated CRH levels stimulate adrenocorticotropic hormone (ACTH) secretion from the pituitary, which independently impairs reproductive function, irrespective of adrenal steroid production.[67] Supraphysiological levels of glucocorticoids are also known to suppress LH responses to GnRH and reduce the effects of estradiol on the uterus.[89]

(2) Physiological Hyperprolactinemia (Table-3)

Hyperprolactinemia is the most common cause of pituitary-associated amenorrhea. These women have reduced LH pulse frequency and reduced LH responsiveness to estrogen, suggesting that GnRH suppression may be a key factor. This is supported by the fact that in hyperprolactinemic amenorrhea women, treatment with pulsatile GnRH results in follicular maturation and ovulation when coupled with a human chorionic gonadotropin trigger. In vitro studies have found that prolactin has a direct inhibitory role on ovarian granulosa cells and thereby directly disrupts ovarian function13,14.

Although most women with hyperprolactinemia have amenorrhea, some have ovulatory menstrual cycles. They do suffer from infertility due to short luteal phase defects15. Therefore, treating hyperprolactinemia may be important even in regularly menstruating women.

A number of physiological causes of hyperprolactinemia and amenorrhea, including pregnancy, do not warrant treatment and, therefore, should be excluded before initiation of treatment.

Table-3; physiological causes of hyperprolactinemia

Pregnancy	Pregnancy is the most common cause of Hyperprolactinemia. Prolactin levels increase during pregnancy and peak at delivery. In women who do not breast feed, prolactin levels usually decrease during the first 72 hours postpartum and normalize by 3 weeks[16,17].
Lactation	Suckling increases prolactin levels, although this usually subsides by 12 weeks due to the postpartum drop in estradiol levels resulting in decreased lactotroph hyperplasia. Similar to prolactin-induced amenorrhea, lactational amenorrhea is associated with reduced LH-pulse amplitude and frequency and reduced LH responsiveness to estrogen[18,19].
Macro-prolactinemia	Predominate prolactin form is 23kD in humans. There is another higher molecular mass form called macroprolactin. It is slowly cleared from circulation and this complex mass is less bioactive plus cause no symptoms. But Hyperprolactinemia that results from macro molecule is evident in ¼ Hyperprolactinemia individuals and they are prone for amenorrhoea[20].

(B) Pathological Secondary Amenorrhoea

(1) Pathological Hyperprolactinemia

Pathological hyperprolactinemia commonly results in amenorrhoea with or without subfertility. Elevated prolactin levels in these cases warrant thorough evaluation. The causes are summarized in Table 4.

Causes of Pathological Hyperprolactinemia

1. Lactotroph Adenoma

Lactotroph adenomas are the most common type of pituitary adenoma that secrete prolactin, making them the leading cause of pathological hyperprolactinemia. The size of the adenoma correlates with serum prolactin levels. These benign lesions cause amenorrhoea through mechanisms similar to those described for physiological hyperprolactinemia.[21]

2. Stalk Disruption

Dopamine, secreted by the arcuate nucleus, exerts tonic inhibition on prolactin secretion. Disruption of the pituitary stalk, which connects the hypothalamus and pituitary, reduces dopamine-mediated inhibition, leading to hyperprolactinemia. Stalk disruption can result from cranial trauma or large seller masses.

3. Primary Hypothyroidism

Thyrotropin-releasing hormone (TRH) stimulates the secretion of both thyroid-stimulating hormone (TSH) and prolactin. Chronic elevation of TRH in primary hypothyroidism leads to hyperprolactinemia, which is reversible with thyroxine replacement therapy.[22,23]

4. Chronic Renal Failure (CRF)

CRF is associated with increased prolactin secretion due to reduced lactotroph responsiveness to dopamine suppression and impaired clearance of prolactin.

However, it is an uncommon cause of secondary amenorrhoea in women.

5. Chest Wall Injury

Chest wall injuries can rarely cause hyperprolactinemia and amenorrhoea. Neurogenic stimulation at the site of injury is believed to be responsible for this effect.[24]

(2) Sellar Mass/Lesions (e.g., Non-Lactotroph Adenomas)

Lesions larger than 1 cm can compress pituitary gonadotrophs or disrupt the stalk, leading to hyperprolactinemia. These include functional or non-functional adenomas, craniopharyngiomas, Rathke's pouch cysts, vascular lesions, or other solid tumors.

(a) Cushing's Disease

Amenorrhoea is a common feature of adrenocorticotropic hormone (ACTH)-secreting adenomas. Up to one-third of affected women experience menstrual irregularities. These patients have markedly elevated serum cortisol levels, reduced serum estradiol, and lower sex hormone-binding globulin (SHBG) levels. Despite elevated adrenal androgens, the amenorrhoea in Cushing's disease is primarily due to GnRH suppression caused by high cortisol concentrations rather than androgen excess.[25]

(b) Acromegaly

Growth hormone (GH) secreting adenomas can cause amenorrhoea and subfertility. These women typically exhibit low LH and estradiol levels, contributing to menstrual irregularities.[26]

(c) Thyrotrophic Adenomas

Thyrotropin (TSH)-secreting tumors are associated with thyrotoxicosis. These tumors can cause elevated SHBG, FSH, LH, and estradiol levels. However, the absence of a mid-cycle LH surge, as seen in physiological cycles, results in anovulation and amenorrhoea in some women.[27]

(d) Infiltrative Conditions

Infective, inflammatory, or malignant infiltrations of the hypothalamic-pituitary axis can induce amenorrhoea. Most cases are secondary to primary breast or lung malignancies.[28]

Examples include:

- Hypophysitis
- Tuberculosis
- Sarcoidosis
- Hemochromatosis

(e) Sheehan's Syndrome

During pregnancy, the pituitary gland enlarges due to estrogen-induced hypertrophy of lactotroph cells. This can compress the pituitary vascular supply. Significant blood loss during pregnancy (e.g., major postpartum hemorrhage) can reduce pituitary perfusion, leading to ischemic necrosis.[29]

Patients with Sheehan's syndrome develop hypopituitarism, manifesting as reduced breast milk production and amenorrhoea. Studies indicate delays in the diagnosis of this condition. Improved labor management protocols aimed at minimizing postpartum hemorrhage have significantly reduced cases of pituitary necrosis secondary to obstetric bleeding.[30]

(f) Cranial Trauma

Traumatic brain injuries and subarachnoid hemorrhages are known to cause hypogonadism and subsequent amenorrhoea.

(g) Adrenal Tumors

Glucocorticoid- and androgen-secreting adrenal tumors can cause amenorrhoea. Excess cortisol suppresses GnRH secretion, while elevated androgens inhibit LH pulse frequency.[31]

(h) Genetic Causes

Several genetic disorders can cause amenorrhoea, often with distinct phenotypic manifestations affecting the hypothalamic-pituitary-adrenal (HPA) or hypothalamic-pituitary-ovarian (HPO) axis. Examples include:

- KAL-1 Gene Mutation: Associated with Kallmann syndrome, an anosmic variant of idiopathic hypogonadotropic hypogonadism.
- KISS1/KISS1R Mutation: Causes idiopathic hypogonadotropic hypogonadism with normal olfactory function (normosmia).

(i) Iatrogenic Causes

Several medical interventions can result in amenorrhoea, including:

- Medication-Induced Amenorrhoea:

GnRH agonists, GnRH antagonists, and opioid use can suppress GnRH secretion or cause hyperprolactinemia. Opioid-induced hypogonadism is primarily mediated by GnRH inhibition. Immunotherapy agents like ipilimumab can induce hypophysitis and subsequent pituitary failure, leading to amenorrhoea.

- Radiation-Induced Hypopituitarism
- Post-Pituitary Surgery

Conclusion

Secondary amenorrhoea often arises from complex neuro-endocrinological disturbances. Evaluation and management require a multidisciplinary approach due to the intricate pathophysiology. Hormone replacement therapy remains a cornerstone of care for addressing vasomotor symptoms and preventing osteoporosis. However, identifying and treating reversible causes is essential to restore gonadotropin function and improve outcomes.

Conflicts of Interest: None

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Original Article

Association of Lifestyle and Reproductive Factors with Age at Natural Menopause among Postmenopausal Women in Gampaha District

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Abstract

Background: Menopause is a natural transition marking the end of a woman's reproductive capacity, defined by the cessation of menstruation for 12 consecutive months without pathological cause or intervention. International studies have shown that lifestyle and reproductive factors may influence its onset. However, research on Sri Lankan women under this topic is limited.

Objectives: To describe the age at natural menopause and lifestyle and reproductive factors of a group of postmenopausal women in Gampaha district and to determine any association between lifestyle and reproductive factors with the age at natural menopause.

Method: A descriptive cross-sectional study with an analytical component was done, with 126 postmenopausal women in a selected Public Health Midwife (PHM) area in Gampaha. Study participants were selected via a multistage cluster sampling method. Data was collected via an interviewer-administered questionnaire which was prepared based on 24-hour dietary recall, International Physical Activity Questionnaire, National Health Interview Survey Tobacco Use Questionnaire, The Alcohol Use Disorders Identification Test and Reproductive History Questionnaire of Women's Health Initiative. Lifestyle factors were studied under four components; dietary factors, physical activities, smoking and alcohol consumption. Age at menarche, parity, total breastfeeding duration and contraceptive usage were assessed under reproductive factors.

Results: The mean age ± standard deviation (SD) at natural menopause was 48.37 (±5.40) years. Premature menopause was experienced by 4.8%. The mean (±SD) daily calorie intake was 961.02 (±233.70) calories. Participants had a mean (±SD) total MET (Metabolic Equivalent of Task) score of 6864.15 (±7574.11) minutes, with 72.2% categorized as having high levels of physical activity. None of the participants were active smokers while 7.9% were exposed to passive smoking. The mean (±SD) age at menarche was 13.73 (±1.32) years, while the mean (±SD) parity was 2.39 (±1.12). A majority (67.5%) had breastfed for over four years in total. Only 35.71% had used contraceptives during their lifetime. No statistically significant associations were observed between the age at natural menopause and lifestyle or reproductive factors.

Conclusion and Recommendations: The mean age at natural menopause of the study

population was 48.37 which aligns with previous studies done among Sri Lankan post-menopausal women under natural conditions. High level of physical activity and a low-calorie intake was observed in the majority emphasizing the need for exploring and improving the nutritional status of postmenopausal women. Lifetime contraceptive use was observed in only about 1/3rd of the participants, highlighting the need for promoting contraceptive practices among women. There was no association between lifestyle or reproductive factors with the natural age of menopause. Future studies with larger and more diverse populations, as well as detailed data collection, are recommended to enable a more comprehensive analysis of associations.

Keywords: Age at menopause, lifestyle, reproductive, diet, physical activity

Background

Menopause is a natural transition marking the end of a woman's reproductive capacity. It can occur as a natural physiological change or as a result of clinical procedures, pathological causes etc. Natural menopause is considered after 12 consecutive months without menstruation and without any other obvious physiological or pathological cause and any clinical intervention. Reduced levels of circulating blood estrogen and loss of ovarian follicular activity are the two main causes of natural menopause. Most women experience natural menopause between the ages of 45 and 55 years as a natural part of biological ageing.(1) The mean age of natural menopause in Sri Lanka is around 51 years.(2) Numerous physiological, hormonal, and psychological changes accompany the menopausal transition and these changes are impacted by sociocultural, psychological and ethnic variables.(3) Postmenopausal women were found to have a high prevalence of menopausal symptoms, which have been demonstrated to lower general health status, increase irritability, depression and bone loss, as well as lower quality of life.

Women experiencing menopause before 40 years of age is termed as premature menopause. (1) Premature menopause results in a longer period of exposure to above mentioned changes. Thus, it is important to identify factors associated with age at natural menopause.

There are several studies revealing association of lifestyle and reproductive factors with age at natural menopause. An Italian cross-sectional study showed a relationship between age at natural menopause and higher parity. (4) However, no correlation was discovered between parity and the age at menopause in a study done in the People's Republic of China. (5) This suggests changes of associated factors in different countries. But available published literature regarding the factors associated with the age at natural menopause in Sri Lankan women is limited.

This study aimed to describe the lifestyle and reproductive factors of postmenopausal women in a semi urban population of Sri Lanka, and explore their association with the age at natural menopause.

Methods

This descriptive cross-sectional study with an analytical component aimed to assess postmenopausal women of ages 40-65, focusing on socio-demographic, lifestyle

and reproductive factors. The study population comprised women residing in Gampaha MOH area using a multi-staged cluster sampling method. Houses of selected streets in the area were visited consecutively and only one eligible participant per household was chosen. Women who met the inclusion criteria (postmenopausal for at least one year, aged 40-65years and residents of the area for over three months) were recruited after obtaining written informed consent.

Data collection was done via a structured interviewer-based questionnaire, covering three sections: socio-demographic factors, lifestyle factors including diet, physical activity, smoking, alcohol consumption and reproductive history (including age at menopause, menarche, parity, and contraceptive use). The questionnaire was developed referring validated instruments; 24-hour dietary recall, the International Physical Activity Questionnaire [6], the National Health Interview Survey Tobacco Use Questionnaire [7], the Alcohol Use Disorders Identification Test [8] and the Reproductive History Questionnaire of Women's Health Initiative. [9] It was translated into Sinhala and Tamil as well. Statistical analysis was conducted using SPSS, with descriptive and analytical tests at a 0.05 significance level.

Ethics approval was obtained from the Ethics review committee, Faculty of Medicine, University of Colombo (MFC/AL/2020/2140).

Results

The study included 135 participants with a response rate of 93.3%. (n=126)

Sociodemographic characteristics

The age of participants ranged from 47 to 65 years with a mean age of 58.96 years (SD= ±4.85). The age distribution of the participants is stated in Table 1.

Table 1: Distribution of the sample by age groups

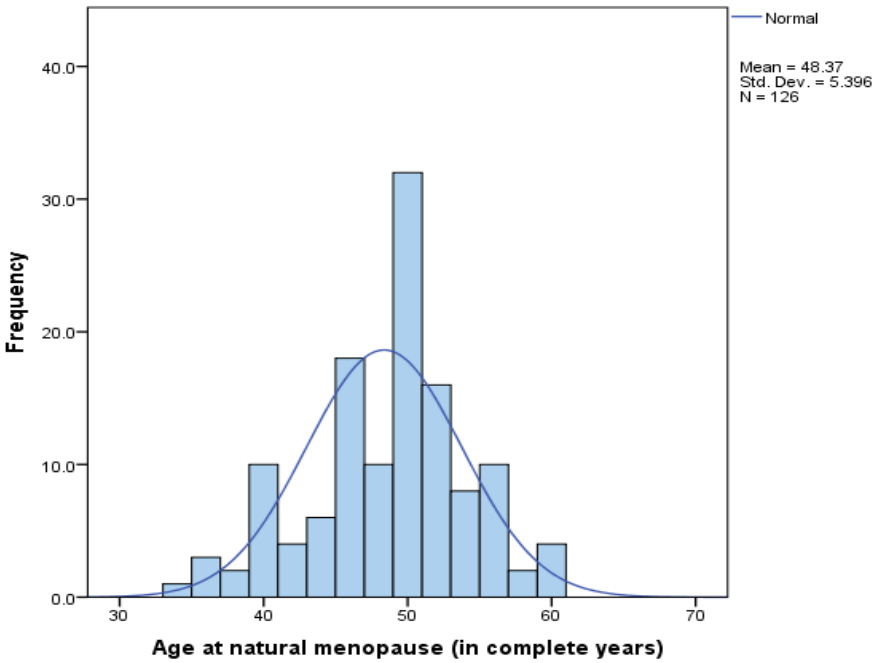
Age groups	Number (n=126)	Percentage (%)
Current age (in complete years**)**		
45-49	6	4.80
50-54	23	18.30
55-59	28	22.20
60-65	69	54.80

Majority of the participants (54.80%, n=69) were in the 60-65 years' age range. All the participants were Sinhalese and the majority of them (77.8%, n=98) were housewives. The majority (62.70%, n=79) had a monthly household income of less than 50,000 LKR.

The age at natural menopause

The age at natural menopause in the participants ranged from 34 to 60 years with the mean age being 48.37(SD= ±5.40) years while both the median and the mode being 50 years. Most participants (38.1%, n=48) had attained natural menopause between the ages of 50-54 years. Figure 1 shows the distribution of age at natural menopause of the participants.

Figure 1: The distribution of age at natural menopause.



The lifestyle factors

In this study dietary factors, physical activities, smoking and alcohol consumption were assessed under the lifestyle factors.

1) Dietary factors

The daily dietary intake of postmenopausal women was assessed in this study. The mean total daily calorie intake of the participants was 961.02 calories. (SD= ±233.70) The median was 962.83 calories while the values ranged between 238.00 and 1681.00 calories. The majority (64.30%, n=81) consumed at least one fruit per day.

Table 2 summarizes the daily consumption of key dietary factors, including total carbohydrate, animal protein, plant protein, total protein, and fat intake.

Table 2; Daily nutrient consumption (in grams)

Dietary categories	Mean (grams)	Minimum (grams)	Maximum (grams)	Standard deviation
Carbohydrate	237.26	109.00	945.13	±174.97
Animal protein	11.13	0.00	27.00	±6.698
Plant protein	15.04	6.0	29.00	±3.477
Protein	26.23	6.0	48.26	±8.59
Fat	17.40	3.83	48.00	±9.62

2) Physical activities

Physical activities per week were studied under engagement in vigorous activities, moderate activities, walking and resting. They are described in MET (Metabolic Equivalent of Task) minutes in Table 3.

Table 3: Engagement in vigorous activities, moderate activities, walking and resting per week

Variable	Number (n=126)	Mean MET	Standard deviation
Vigorous activities	21	2034.29	±6257.09
Moderate activities	126	3371.43	±2124.42
Walking	116	1458.44	±1665.38
Resting	126	1543.33	±1044.89
Total activities	126	6864.15	±7574.11

All the participants had engaged in moderate and resting activities. Majority of the participants (92.1%, n=116) had engaged in walking activities and only twenty-one participants (16.67%) had engaged in vigorous physical activities.

The total MET minutes per week were categorized into three groups: low, moderate and high. Majority of the participants (72.2%, n=91) had engaged in high physical activities according to MET minutes' categorization.

3) Smoking and alcohol consumption

There were no active smokers among the participants of the study. However, there were 10
(7.9%) passive smokers out of them 4 (40%) had been exposed to the smoke for one to two hours per day and six (60%) had been exposed for 2-5 hours per day.

Of the participants, only one (0.8%) had consumed alcohol. And that was 1-2 glasses per time with a frequency less than once a month.

The reproductive factors

The mean age at menarche was 13.73 years (SD= ±1.32). It ranged from 11 to 17 years and the mode 13 years. Twelve participants (9.5%) had experienced irregularities in menstruation in their lifetime, with 10 having irregularities throughout their reproductive ages.

All participants had been pregnant at least once. The mean age at first pregnancy and last pregnancy are 26.79 years (SD= ±5.70) and 32.89 years (SD= ±5.16) respectively. Most had 3 pregnancies (n=42, 33%), and had delivered 2 live births (n=50, 38.1%). Every participant has at least one live birth while 38 participants (31.20%) have had at least one miscarriage. Only two (1.59%) had attempted to conceive without success for more than a year, but neither of them has consulted a doctor and is therefore unaware of the reason.

All participants have breastfed their children. The mean ages at first and last breastfeeding was 26.98 years (SD= ±6.31) and 34.60 years (SD= ±5.28) respectively. The majority of participants (67.50%, n=85) had breastfed for more than four years.

Only 44 out of 126 participants (34.90%) reported using one contraceptive method during their lifetime, while three participants (2.38%) reported using two methods. The most commonly used contraceptive method among participants was oral contraceptive pills (34.04%, n=16). Additionally, the mean duration of oral contraceptive pill usage among those who used it was 38.25 months (SD= ±23.17 months). Table 4 describes the reproductive factors of the participants.

Table 4 : Distribution of reproductive factors among the participants.

Category	Number (n=126)	Percentage (%)
1) Age at menarche		
Early menarche (<12 years)	2	1.60
Normal menarche (12-15 years)	108	85.70
Late menarche (>15 years)	16	12.70
2) Gravidity		
1	16	12.70
2	37	29.40
3	42	33.33
4	21	16.70
5	10	7.90
3) Live births		
1	25	19.80
2	50	39.70
3	34	27.00
4	15	11.90
5	2	1.60
4) Miscarriages		
0	88	69.80
1	32	25.40
2	4	3.20
3	2	1.60
5) Total breastfeeding duration		
4-6 months	2	1.60
7-12 months	8	6.30
13-23 months	16	12.70
2-4 years	15	11.90
> 4years	85	67.50

Association of lifestyle factors with the age at natural menopause

1) Dietary factors

The study aimed to explore the relationship between various dietary intakes per day and the age at natural menopause among this group of participants. The results presents Pearson correlation coefficients (r values) and their significance (p values) levels for each dietary component were analyzed. These correlations were not statistically significant, indicating no statistically significant relationship.

Fruit consumption and its association with the age at natural menopause was done among the 126 participants. The data were categorized based on daily fruit intake: those not consuming fruits and those consuming at least one fruit per day. A t-test was conducted to compare the mean age at natural menopause between the two groups. The t-test results indicate no significant difference in the age at natural menopause between the two groups (p=0.907).

Table 5: Association of daily dietary intake with age at natural menopause

Dietary component	Pearson correlation	Significance (2-tailed)
Total carbohydrate intake per day	-0.046	0.613
Total animal protein intake per day	0.095	0.291
Total plant protein intake per day	0.019	0.835
Total protein intake per day	0.062	0.493
Total fat intake per day	-0.087	0.333
Total calorie intake per day	0.090	0.319

2) Physical activities

The participants were divided into two groups: “moderate to low physical activity” (less than 3000 MET minutes per week, combining low and moderate) and “high physical activity” (more than 3000 MET minutes per week). There was no significant difference (p= 0.378) between the natural age at menopause in “moderate to low physical activity” physical activity group and “high” physical activity group using an independent sample t-test.

3) Smoking and alcohol consumption

There were not enough numbers to analyze any association between smoking and alcohol consumption with the age at natural menopausal because there were no active smokers, only ten passive smokers and only one-woman consuming alcohol among the participants.

Association of reproductive history with the age at natural menopause

Any associations with the age at natural menopause with variables in the reproductive history of women were calculated using the independent sample t test and the Pearson’s correlation coefficient. Age at natural menopause did not show any significant association with age at menarche (p=0.115), total breastfeeding duration (p=0.182) and the

use of oral contraceptives (p=0.850) using independent sample t-tests. There was also no significant correlation between parity and age at natural menopause using the Pearson correlation coefficient (r= -0.078, p=0.383).

Discussion

The mean age at natural menopause among participants in this study was 48.37 years (SD = ±5.40), which aligns with previous findings from a Sri Lankan study reporting a mean age of 48.35 years.[10] Compared to international studies, Indian women had a slightly lower mean menopause age of 46.60 years,[11] while women in the United Kingdom had a mean age of 47.80 years,[12] also on the lower side. In contrast, Korean women had a slightly higher mean menopause age of 49.30 years,[13] potentially influenced by genetic, lifestyle, or socioeconomic factors. Notably, 4.8% of participants experienced premature menopause (before 40 years), a condition associated with increased risks of cardiovascular disease and osteoporosis. This finding highlights the importance of targeted healthcare interventions to address the unique health challenges faced by this group.

Dietary analysis revealed significant nutritional deficiencies among postmenopausal participants. The mean daily caloric intake was below the recommended levels for women in this age group, reflecting inadequate energy intake.[14] Carbohydrates were the primary macronutrient consumed, reflecting typical dietary patterns, but variability in intake was high. Excessive carbohydrate consumption among some participants could increase the risk of insulin resistance, especially in this high-risk group. Protein and fat intake were consistently below recommended levels, raising concerns about insufficient essential fatty acids crucial for cardiovascular health and hormonal balance. Moreover, inadequate fruit consumption was evident, with 35.7% of participants reporting no daily fruit intake. This poses a risk of nutrient deficiencies, weakened immune function, and chronic diseases. Overall, poor nutrient and caloric intake among postmenopausal women could adversely impact health outcomes, emphasizing the need for nutritional interventions.

Physical activity levels among participants were generally high, primarily due to moderate activities like walking and household or agricultural tasks. Vigorous activities were infrequent, consistent with earlier findings that household and occupational tasks significantly contribute to physical activity levels.[15] The use of the International Physical Activity Questionnaire (IPAQ) provided useful insights; however, its MET minute categorization may not fully capture Sri Lankan activity patterns. Reporting biases related to age, occupation, and social class may have influenced the results.[15]

Reproductive health findings were consistent with prior research. The mean age at menarche was 13.73 years, with 85.7% of participants reporting normal menarche. This age aligns closely with international pooled data reporting a mean age of 13.2 years.[16] The mean parity was 2.39, reflecting cultural norms favoring smaller families in Sri Lanka.[17] Miscarriages were reported by 31.2% of participants, highlighting the need for awareness and support related to pregnancy

loss. Breastfeeding practices were prevalent, with 67.5% of participants breastfeeding for over four years, in accordance with WHO recommendations. However, only 35.71% reported contraceptive use, primarily oral contraceptive pills, while 65.1% had never used contraception. These findings reflect cultural barriers and limited awareness regarding family planning, consistent with prior studies. [17]

Contrary to expectations, no significant associations were observed between dietary intake carbohydrates, proteins, fats, and calories and the age at natural menopause. These findings differ from studies like the Nurses' Health Study, which identified links between dietary patterns and menopause timing. Differences may stem from variations in dietary quality and long-term patterns. The use of a 24-hour dietary recall, while providing detailed data, limited the ability to assess habitual dietary patterns. The absence of a food frequency questionnaire (FFQ) was due to concerns about recall bias and participant fatigue, which may have restricted the depth of dietary assessments.

Similarly, no significant associations were identified between physical activity levels and the age at natural menopause. This contrasts with studies conducted in China and Japan, which reported differing relationships between physical activity and menopause timing. [18,19] These differences may be attributed to genetic predispositions or variations in activity types, such as manual labor versus structured or leisure-time exercise.

Reproductive factors, including age at menarche, parity, breastfeeding duration, and contraceptive use, also showed no significant association with the age at natural menopause. For example, early menarche (≤15 years) was not significantly correlated with menopause timing ($p = 0.115$), contrasting with findings from international pooled data. [16] Similarly, parity and breastfeeding duration showed no significant relationships.

The study has several strengths, including the use of validated international questionnaires and a comprehensive assessment of lifestyle and reproductive factors. However, there are notable limitations. The small sample size (126 participants) and cross-sectional design limit the generalizability and ability to infer causality. The exclusive focus on Sinhalese women restricts ethnic diversity, while the reliance on 24-hour dietary recall may not reflect long-term dietary habits. Additionally, recall bias may have influenced data on physical activity and contraceptive use.

Future research should employ larger, prospective cohort studies with diverse populations to explore long-term dietary and lifestyle patterns. Public health initiatives should focus on addressing nutritional deficiencies, improving family planning awareness, and supporting self-employment opportunities to enhance the health and well-being of postmenopausal women.

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Review Article

Pelvic Reconstructive Surgery in Urogynaecology

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Abstract

Pelvic organ prolapse (POP) is a prolapse of one or more of the anterior vaginal wall, posterior vaginal walls, uterus (cervix), and vaginal vault (after hysterectomy). Pelvic reconstructive surgery plays a key role in management of POP. This is subclassified in to different classifications based on the anatomical position and the approach of the surgery. Uterine preserving sugery for uterine prolapse has become more popular during recent decades.

Introduction

Pelvic organ prolapse (POP) is a herniation of the pelvic organs including uterus, vaginal cuff, bladder, urethra, small or large bowel, rectum, and associated vaginal segments due to weakness or defect in the pelvic floor. POP is defined by the International Urogynecological Association (IUGA) and International Continence Society (ICS), in Joint Report on Terminology, as the "Descent of one or more of the anterior vaginal wall, posterior vaginal wall, the uterus (cervix) or the apex of the vagina"[1]. Prevalence of pelvic floor dysfunction is estimatd as 30-50% of the female population.

POP is sub-claasified as anterior compartment prolapse, apical compartment or uterine prolapse, and posterior compartment prolapse. Different types of pelvic organ prolapse includes;

- Anterior compartment – cystocele, urethrocystocele
- Apical compartment – uterine prolapse, vaginal vault prolapse (post-hysterectomy)
- Posterior compartment – enterocele, rectocele

POP can be managed with conservative management and surgical management. Surgical management of POP includes pelvic reconstructive surgeries and pelvic obliterative surgeries. Pelvic reconstrctive surgery is the restoration of the pelvic floor support. Main goals of pelvic reconstructive surgery includes; repairing all the defects, restoration of normal anatomy, maintaining normal vaginal function, improvement of symptoms related to POP, improvement of QOL, and prevention of recurrence. Life time risk of surgery for POP is 11% by age of 80 years[2]. Reoperation risks after previous surgery is 13-56%.

Reconstructive surgery for POP is sub-classifed in to different classifications based on the anatomical posisiton of the defect and the approach of the surgery.

Classification based on the anatomical position is done as anterior compartment surgeries, posterior compartment surgeries and apical compartment surgeries. Classification based on the surgical approach is done as transvagianl approach, transabdominal (open) approach, laparoscopic and robotic (da Vinci robotic surgery) approach. Choice of the surgery will be based on the type and severity of POP, surgeon's skills, surgical experience, and patient's preference.

Clinical Presentation

Common presentations of POP icludes bulge in the perineum and palpable mass in the perineum[3]. It may also present or associte with urinary symptoms, bowel symptoms, vaginal discharge, PV bleeding, ulceration, and perineal discomfort. Urinary symptoms may include voiding symptoms like hesitancy, poor stream, retention and incontinance. Bowel symptoms may include constipation and feacal soiling. Common sexual symptoms may include lack of sexual desire and superficail dyspareunia. Some women may have significant effects of quality of the life, increasing the risk of depression and anxiety.

Diagnosis of POP

Dignosis of POP is by clinical examination usually at dorsal position or some times at standing position. Valsalva manoeuvre is used to demonstrate maximum descent. Each compartment is asssessed seperately. Different classification systems are used to stage the degree of descent. The POP-Q grading system is the widely accepted, objective and a reproducible system, adopted by the International Continance Society (ICS), which classifies prolapse into grades 0 to grade IV[4]. The quantitative assessment of the anterior, apical, and posterior vaginal supports, the genital hiatus, the perineal body, and the vaginal length helps surgical planning when planing the management. The degree of the prolpse in each compartment is measured seperately compared to the level of hymen. Co-existing stress urinary incontince should be assessed during the examination.

The Modified Oxford Scale (MOS) is used to assess pelvic floor muscle strength on a scale of 0 to 5; 0 = no contraction; 1 = minor muscle 'flicker'; 2 = weak muscle contraction; 3 = moderate muscle contraction; 4 = good muscle contraction, and 5 = strong muscle contraction[5]. Digital assessment of pelvic floor muscle contraction was performed by inserting the index and middle fingers approximately 4cm into the vagina and palpating the puborectalis muscle at each side of the vagina during the contraction.

Surgeries for POP

1. **Colporrhaphy (Native tissue repair)**

Colporrhaphy is a minimally invasive traditional surgical procedure which repair the pelvic floor defect. This consists of removal of excessive over-lying vaginal wall, restoration and support of the prolapsed pelvic organ, and plication of the under-lying supportive fascia. This is sub-classified as anterior colporrhaphy and posterior colporrhaphy based on the anatomical location of the surgery. However, this may asscoiated with increaed recurrence risk of 30%.

Anterior colporrhaphy / Cystocele repair - is the repair of anterior vaginal wall defects. This is repaired by excision of excessive vaginal wall and central plication of fibromuscular layer of anterior vaginal wall. Success rate of the procedure is 80-100% at 1 year follow-up and 37-57% at long-term follow-up[6]. Main drawback of the procedure is high recurrence rate.

Posterior colporrhaphy / Rectocele repair - is the repair of posterior vaginal wall defect which involves plication of the posterior vaginal muscularis and rectovaginal septum or medial aspect of levator ani muscle in the midline. It also narrows the diameter of the vaginal canal, traditionally up to three-fingerbreadth genital hiatus. Usually, the procedure is performed with perineorrhaphy together. Posterior colporrhaphy has an anatomic cure rate of 76% to 96%[7].

2. Transvaginal mesh repair

Transvaginal mesh repair is a controversial topic and is not widely practised due increased rate fo drawbacks including vaginal mesh exposure, bladder or urethral erosion, dyspareunia, and significant pelvic pain. Synthetic mesh repair was first approved by the United States Food and Drug Administration (FDA) in year 2002. Afterwards, it was used commonly due to its "user-friendly" format. Currently it's use is not recommended by FDA, and the United Kingdom's Medicines and Healthcare products Regulatory Agency (MHRA)[8,9].

However, recently puplished Cochrane Library meta-analysis showed non-absorbable mesh repair reduces the risk of anatomical recurrence, repeat surgery for prolapse recurrence, as well as patient awareness of prolapse after surgery, when compared to non-mesh repair[10]. Conversely, the recently published "PROlapse Surgery: Pragmatic Evaluation and randomised Controlled Trials" did not show significant difference in patient satisfaction, quality of life, and anatomic success when comparing synthetic mesh and native tissue repair[11].

In summary, the decision on mesh repair is stilll remain highly controversial. Traditional native tissue repair remains a mainstay of POP surgery for uncomplicated cases. Transvaginal mesh repair can be considered as second-line procedure, or for patients with significant grade of prolapse who are at a higher risk of recurrence, and these procedures should only be performed by a well-trained and skilled surgeon.

3. Vaginal vault suspension

Apical compartment prolpase can be traeted with apical suspension techniques which include sacrospinous ligament suspension and uterosacral ligament suspension. Currently avaialble evidance does not demonstrate superiority of either technique. Vaginal vault suspension techniques are considered as low risk and most siutable for elderly and sexually non-active patients who are not suitable for abdoinal vault suspension.

Sacrospinous ligament suspension / Scaropinous fixation (SSF) – is the most commonly used transvaginal procedure and can be done as unilateral or bilateral procedure. Right-side is more preferred. The vaginal vault is anchored to the sacrospinous ligament, restoring its support. SSF is associated with increased risk of postoperative dyspareunia due to

deviation of the vagainal angle, increased buttock pain (30%), and higher recurrence rate (30%). Objective cure rate is 67-97%.

Uterosacral ligament suspension – the vaginal vault is anchored to the uterosacral ligament. However, this procedure is associated with increased risk of ureteric obstruction in 3.7% of the cases due to its close proximity to ureter[12].

4. Abdominal sacrocolpopexy (ASC) / scarohysteropexy (ASH)

ASC / ASH is used in isolated apical compartment proplase or combined apical and anterior comparment prolpase[13]. During this procedure, the vaginal vault or cervix is anchored to the anterior surface of the sacral promontory, by means of a mesh (usually a synthetic polypropylene mesh). The procedure can be done as a open lapartomy procedure, laparoscopic procedure and robotic assisted procedure.

Main benefit of ASC is significantly lower postoperative dyspareunia and considred as the choice of option for sexually active women[14]. The success rate of the procedure is 90% at 5 years, and 74% at 13.7 years.

Laparoscopic ASC has the advantages of shorter hospital stay, early recovery, less post-operative pain and decreased blood loss[15]. Recent randomised control trial showed that open and laparoscopic ASC were equivalent for apical prolapse repair, but that the laparoscopic approach was inferior with regard to concomitant anterior compartment prolapse[16].

The da Vinci Surgical System is a robotically assisted surgical device used for ASC in robotic surgery and approved by FDA in year 2000. Main advantages of robotic assisted surgery over laparoscopic ASC includes better depth perception due to 3D vision, better ergonomics, fast learning curve and more natural surgical feel[17]. The technique is more similar to open repair but with the decreased operative morbidity associated with laparoscopy. However, cost and the operative time for robotic surgery was significantly higher.

5. Vaginal hysterectomy and repair

Vaginal hysterectomy with POP repair is the traditional mainstay of primary POP surgery due to easy access to pelvic ligaments and tissues for vaginal suspension, especially in uterine prolapse. Vaginal hysterectomy is typically accompanied by vaginal vault suspension by either uterosacral ligament suspension (USLS) or sacrospinous ligament fixation (SSF). Vaginal hysterectomy is considered superior to abdominal or laparoscopic hysterectomy due to shorter duration of hospital stay (WMD 1.0 day, 95% CI: 0.7 to 1.2 days), early return to normal activities (WMD 9.5 days, 95% CI: 6.4 to 12.6 days), and fewer unspecified infections or febrile episodes (OR 0.42, 95% CI: 0.21 to 0.83)[18].

However, uterine presserving surgery was introduced in recent decades and became more popular. Systematic reviews indicate that uterine preserving surgery is associated with lower perioperative risks than vaginal hysterectomy, with comparable rates of POP cure[19]. Different options available for uterine preservation surgery include the Manchester Fothergill's operation, sacral hysteropexy (abdominal, laparoscopic or robotic with or without mesh), uterosacral ligament hysteropexy, sacrospinous

hysteropexy (with or without mesh), and colpocleisis[20].

6. Colpocleisis

Colpocleisis is considered as an pelvic obliterative surgery. LeFort colpocleisis procedure involves removal of strips of anterior and posterior vaginal epithelium, leaving a small strip of lateral epithelium on each side and suturing of the remaining vaginal epithelium to creat a outlet for cervical or uterine bleeding or drainage. This is considered as the last resort if all other options are not applicable.

Conclusion

Pelvic reconstructive surgeries play key role in the management of POP. Colporrhaphy is considered as the management of choice for mild POP, after failure of conservative management. Transvaginal mesh repair for POP is highly contraversial and can be considered in selected cases, especially in recurrent POP following failing of traditional colporrhaphy. ASC is the treatment of choice for apical compartment prolapse, especailly in sexually active women. Laparoscopic ASC is considered superior over open ASC due to early recovery, early return to work and minimal blood loss. Robotic assisted ASC is very costly and time consuming. Vaginal hysterectomy with POP repair is performed in uterine prolapse but carries higher perioperative and postoperative risks. Uterine preserving surgeries become more popular in recent decades due to lower risk of complications compared to traditional vaginal hysterectomy.

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Reference: 1. Amy JJ. Femoston®: Effects on bone and quality-of-life. Eur Menop J 1995;2(4) (Suppl):16–22. 2. Stevenson JC, Durand G, Kahler E, et al. Oral ultra-low dose continuous combined hormone replacement therapy with 0.5 mg 17ß-oestradiol and 2.5 mg dydrogesterone for the treatment of vasomotor symptoms: Results from a double-blind, controlled study. Maturitas 2010;67:227–232. 3. Cieraad D, Conradt C, Jesinger D, et al. Clinical study comparing the effects of sequential hormone replacement therapy with oestradiol/ dydrogesterone and conjugated equine oestrogen/norgestrel on lipids and symptoms. Arch Gynecol Obstet 2006;274:74–80.Image for representation purpose only, not of actual patient

Abbreviated Prescribing Information Estradiol and Dydrogesteron Tablets Femoston ® conti 1/5 mg Combipack of Estradiol and Dydrogesterone Tablets Femoston 1/10 mg Combipack of Estradiol and Dydrogesterone Tablets Femoston 2/10 mg LABEL CLAIM: Each film coated tablet contains: Estradiol (as hemihydrate) Ph Eur .....1 mg Dydrogesterone IP.....5mg Femoston ® 1/10 mg Each of 14 white colour tablet contains: Estradiol (as hemihydrate) Ph Eur .....1 mg Other each 14, grey colour film coated tablets contains: Estradiol (as hemihydrate) Ph Eur .....1 mg Dydrogesterone IP.....10 mg FemostonTM 2/10 mg Each of 14 white colour tablet contains: Estradiol (as hemihydrate) Ph Eur .....2 mg Other each 14, grey colour film coated tablets contains: Estradiol (as hemihydrate) Ph Eur .....2 mg Dydrogesterone IP .....10 mg INDICATION: For Continuous combined: FemostonTM conti 1/5 mg Hormone replacement therapy (HRT) for oestrogen deficiency symptoms in postmenopausal women at least 12 months since last menses. For Continuous sequential: FemostonTM 1/10 mg Hormone replacement therapy (HRT) for oestrogen deficiency symptoms in postmenopausal women at least 6 months since last menses.For Continuous sequential: FemostonTM 2/10 mg Hormone replacement therapy (HRT) for oestrogen deficiency symptoms in postmenopausal women at least 6 months since last menses. All formulations: Prevention of osteoporosis in postmenopausal women at high risk of future fractures who are intolerant of, or contraindicated for, other medicinal products approved for the prevention of osteoporosis. Elderly population: The experience in treating women older than 65 years is limited. DOSAGE AND ADMINISTRATION: For oral use FemostonTM conti 1/5: Continuous combined. The oestrogen and the progestogen are given every day without interruption. One tablet to be taken daily for a 28 day cycle. FemostonTM 1/10 and FemostonTM 2/10: Continuous sequential. The oestrogen is dosed continuously. The progestogen is added for the last 14 days of every 28 day cycle, in a sequential manner. For FemostonTM 1/10 and FemostonTM 2/10: Treatment commences with one white tablet daily for the first 14 days followed by one grey tablet daily for the next 14 days, as directed on the 28 day calendar pack. All Formulations: Femoston should be taken continuously without a break between packs. For initiation and continuation of treatment of postmenopausal symptoms, the lowest effective dose for the shortest duration should be used. For FemostonTM 1/10: In general, sequential combined treatment should start with FemostonTM 1/10. For FemostonTM conti 1/5: Continuous combined treatment may be started with FemostonTM conti 1/5 depending on time since menopause and severity of symptoms. All Formulations: Depending on the clinical response, the dosage can subsequently be adjusted. FemostonTM can be taken irrespective of food intake. CONTRAINDICATIONS: Known past or suspected breast cancer. Known or suspected oestrogen-dependent malignant tumours (e.g. endometrial cancer). Known or suspected progestogen-dependent neoplasms. Undiagnosed genital bleeding. Untreated endometrial hyperplasia. Previous or current venous thromboembolism (deep venous thrombosis, pulmonary embolism). Known thrombophilic disorders (e.g. protein C, protein S or antithrombin deficiency). Active or recent arterial thromboembolic disease (e.g. angina, myocardial infarction). Acute liver disease, or a history of liver disease, as long as the liver function tests have failed to return to normal. Porphyria. Known hypersensitivity to the active substances or to any of the excipients. WARNINGS & PRECAUTIONS: For the treatment of postmenopausal symptoms, HRT should only be initiated for symptoms that adversely affect quality of life. In all cases, a careful appraisal of the risks and benefits should be undertaken at least annually and HRT should only be continued as long as the benefit outweighs the risk. If any of the following conditions are present, have occurred previously, and/or have been aggravated during pregnancy or previous hormone treatment, the patient should be closely supervised such as leiomyoma (uterine fibroids) or endometriosis, risk factors for thromboembolic disorders, risk factors for oestrogen dependent tumours, e.g. 1st degree heredity for breast cancer, hypertension, liver disorders (e.g. liver adenoma), diabetes mellitus with or without vascular involvement, cholelithiasis, migraine or (severe) headache, systemic lupus erythematosus, history of endometrial hyperplasia, epilepsy, asthma, otosclerosis. Therapy should be discontinued in case a contraindication is discovered and in the following situations such as jaundice or deterioration in liver function, significant increase in blood pressure, new onset of migraine-type headache and pregnancy. PREGNANCY & LACTATION FemostonTM is not indicated during pregnancy. If pregnancy occurs during medication with FemostonTM treatment should be withdrawn immediately. The results of most epidemiological studies to date relevant to inadvertent fetal exposure to combinations of oestrogens with progestogens indicate no teratogenic or foetotoxic effect. There are no adequate data from the use of estradiol/dydrogesterone in pregnant women. Lactation: FemostonTM is not indicated during lactation. ADVERSE REACTIONS: The most commonly reported adverse drug reactions of patients treated with estradiol/dydrogesterone in clinical trials are headache, abdominal pain, breast pain/tenderness and back pain. Issued on: Date (12/Feb/2016) Source: Prepared based on full prescribing information (version 2) dated 25/May/2014

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