Original Research Article

Indian Journal of Emergency Medicine
Volume 4 Number 3, July - September 2018
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.4318.16

A Study on Clinical Profile of Premenopausal and Postmenopausal Women

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Received on 02.07.2018,
Accepted on 17.07.2018

Abstract

Menopausal symptoms are climacteric symptoms, hot flushes due to sudden change in the hormones levels and is not seen in women who are chronically hypo estrogenic. The term "hot flush" is descriptive of sudden onset of reddening of skin overhead, neck and chest accompanies by feeling of intense body heat and concluded by sometimes-profuse perspiration. The duration varies from few seconds to several minutes, rarely for an hour. Only 100 postmenopausal women and 100 premenopausal women and 100 premenopausal women were included after a detailed medical history and were selected on the basis of the following inclusion and exclusion criteria. The FBS of premenopausal group was 78.67 mg/dl and of postmenopausal group was 79.47 mg/dl. The PPBS of premenopausal was 97.3 mg/dl and of postmenopausal group was 97.25 mg/dl. There was no statistical difference in FBS, PPBS, between the two groups.

Keywords: FBS; Premenopausal; Postmenopausal.

Introduction

Menopause is an estrogen deficient state. But unlike other hormone deficient state, menopause is not a disease. In young women estrogen production is high, serum lipids are normal, but after menopause abnormal lipid levels and increased incidence of CHD show a possible relationship among estrogen normal lipid levels and a relative immunity to coronary heart disease [1].

During climacteric, ovarian activity declines. Climacteric indicates the period of time when the women passes from the reproductive stages of life through the perimenopausal transition and the menopause to the postmenopausal years [2]. After menopause progesterone secretion decreases. Initially, ovulation fails, no corpus luteum forms and no progesterone is secreted by the ovary. Later, graffian follicles also fail to develop, estrogenic activity is reduced, and endometrial atrophy leads to amenorrhea.

Cessation of ovarian activity and a fall in the estrogen level as well as inhibin level cause a rebound increase in the secretion of FSH, as much as 50 fold and LH 3-4 fold by the anterior pituitary gland. Prior to menopause, estradiol levels range from 40 to 400 pg/ml. However, even with cessation of ovarian function, levels of estradiol may remain as high as 100 pg/ml [3]. With further advancing years, gonadotrophin activity of the pituitary gland also ceases, a fall in FSH level eventually occurs. Androstenedione produced by the adrenal and ovary and is aromatized to estrogen primarily by muscle and adipose tissue [4]. Obese women have an increased levels of circulating estrogens, and unopposed estrogens places them at an increased risk for endometrial carcinoma. In contrast these women have decreased level of circulating estrogens and are increased risk for developing osteoporosis [5].

There is 50% reduction in androgen production and 66% reduction in estrogen at menopause. The estrogen level may remain low at 10 to 20 ph/ml. Estrogen level of over 40pg/ml exerts bone and cardiotrophic effect, but the level below 20pg/ml may predispose to osteoporosis and ischaemic heart disease. After menopause there is loss of ovarian function. This result in adverse changes in glucose and insulin metabolism, body fat distribution, coagulation, fibrinolysis of vascular endothelial dysfunction [6].

Menopausal symptoms are climacteric symptoms, hot flushes due to sudden change in the hormones levels and is not seen in women who are chronically hypo estrogenic. The term "hot flush" is descriptive of sudden onset of reddening of skin overhead, neck and chest accompanies by feeling of intense body heat and concluded by sometimes-profuse perspiration. The duration varies from few seconds to several minutes, rarely for an hour. Frequency may be rare to recurrent every few minutes. Flushing is more frequent and severe at night or during stress.

Neurotic and psychotic symptoms include depression, nervousness, irritability, insomnia, and headache. The overall "quality of life" reported by women can be improved by better sleep and alleviation of hot flushing. Estrogen therapy as reported to have a more powerful impact on women's well being beyond the relief ofsymptoms such as hot flushes. In elderly depressed women, improvements in response to fluoxetine were enhanced by addition of estrogen therapy [7,8].

Urinary symptoms include dysuria, urgency and recurrent UTI. In addition genuine stress incontinence may be related to estrogen deficiency. Urethral shortening associated with postmenopausal atrophic changes may result in urinary incontinence. Estrogen therapy may improve or cure stress incontinence in more than 50% of treated women presumably by exerting direct effect on urethral mucosa [9].

Methodology

In this study total 200 subjects were included, who were divided into two groups, 100 premenopausal and 100 postmenopausal groups. The study was done by obtaining serum sample from study group and controls, from randomly selected postmenopausal and premenopausal women of similar height and weight of general population of city. Only 100 postmenopausal women and 100 premenopausal women and 100 premenopausal women were included after a detailed medical history and were selected on the basis of the following inclusion and exclusion criteria. Some subjects were on mixed diet and some were vegetarians. Postmenopausal state was considered in women who had at least 12 months amenorrhea.

They were recruited in the research process after having their due consent on printed performa. On the day of the enrolment, the subjects were narrated fully about the protocol of the study.

Inclusion criteria

- Females aged above 50 years with amenorrhoea of more than 1 year.
- No pre-existing medical disease (diabetes mellitus, hypertension, hypothyroidism, chronic illness).
- No history of premature menopause or surgical menopause.
- No history of steroid hormone intake.

Exclusion criteria

- Obese women with body mass index more than 25, to minimize the confounding effect on lipid concentration.
- Those who have undergone premature menopause before 45 years.
- Those who had surgical menopause.
- Patients on anti-inflammatory drugs, antidepressants thyroid hormone.
- Patients with history of liver disease, alcohol consumption, smoking, hypertension and diabetes mellitus.
- Those who are on medication known to influence lipid metabolism (e.g. Sex steroids).

Results

The mean age group of premenopausal women was 33.68 yrs and of postmenopausal group was 55.50 yrs.

The [mean SD] BMI of premenopausal was 21.64 kg/m2 and of postmenopausal group was 21.34 kg/m2. There was no statistical difference in BMI between the two groups (Table 1).

The FBS of premenopausal group was 78.67 mg/dl and of postmenopausal group was 79.47 mg/dl. The PPBS of premenopausal was 97.3 mg/dl and of postmenopausal group was 97.25 mg/dl. There was no statistical difference in FBS, PPBS, between the two groups (Table 2).

Table 1: age of premenopausal and postmenopausal group.

Parameter	Premenopausal group [mean SD]	Postmenopausal group [mean SD]	'z' value	'p' value	Significance
Age (years)	33.68 6.36	55.502.22	32.83	<0.0001	Hs

Table 2: BMI of premenopausal and postmenopausal group.

Parameter	Premenopausal group [mean SD]	Postmenopausal group [mean SD]	'z' value	'p' value	Significance
BMI (kg/m2)	21.64 2.19	21.34 2.19	0.96	>0.05	NS

Table 3: FBS and PPBS of premenopausal and postmenopausal group

Parameter	Premenopausal group [mean SD]	Postmenopausal group [mean SD]	'z' value	'p' value	Significance
FBS (mg/dl)	78.67	79.47	0.96	>0.05	NS
PPBS 9mg/dl)	97.34	97.25	0.10	>0.05	NS

Table 4: Background Characteristics

Parameter	Premenopausal group [mean SD]	Postmenopausal group [mean SD]	Significance (z and p value)
Age (yrs)	33.68	55.50	z=32.3 p<0.0001
BMI (Kg/m2)	21.64	21.34	z=0.96 p>0.05
FBS(mg/dl)	78.67	79.47	z=0.98 p>0.05
PPBS(mg/dl)	97.34	97.25	z=0.10 p>0.05
Inference	FBS and PPBS are statistically similar in both the groups. BMI is also statistically similar (P>0.05). The samples are matched with respect to FBS, PPBs and BMI.		

Discussion

The effect of endogenous and exogenous hormones in females is potentially a major factor in determining cardiovascular risk. Permenopausal females have a considerably lower incidence of cardiovascular disease than postmenopausal females of the same age.

Estrogen appears to increase triglyceride levels and increases LDL catabolism as well as lipoprotein receptors numbers and activity, resulting in decreasing LDL levels. The increase in HDL levels particularly the HDL2 subfraction, is due to the consequence of the inhabitation of hepatic lipase activity, which converts HDL2 to HDL3 [10].

Estrogen is also an antioxidant. Estradiol directly inhibits LDL oxidation in response to copper and decreases the overall formation of lipid oxides. In addition esterogen may generate circulating antioxidants (tocopherols and B-carotene) and preserve these antioxidants whthin LDL particles. This will prevent the lipid peroxidation and accumulation of LDL cholesterol inside the macrophages and the smooth muscle cells [11].

Several aspects of coronary risk in females include the stronger role of diabetes mellitus, hypertriglyceridaemia and HDL compared to men. Hence, carefull attention to these issues holds the promise of reduction of cardovaschular morbidity in adult women. Dyslipidemia is a modifiable risk factor for cardiovascular disease in postmenopausal women, early treatment of which will reduce the risk of cardiovascular disease in them [12].

Coronary artery disease is the leading cause of death in postemenopausal women. Natural menopause confers an increase in CAD risk. Blood lipids are an important risk factor for atherosclerosis in women. The main aim of this study is to study the effect of menopause on plasma lipid cholesterol.

Our study group consisted of 100 pre-menopausal and 100 post menopausal women with the average age of 33 years in premenopausal group and 55 years in postmenopausal groups.

In our study, most of the cases have the normal body mass index, and they are free from hypertension, diabetes mellitus, heart and kidney disease.

The BMI of premenopausal women was 21.64 and of postmenopausal women was 21.342.28. Kg/m2

The FBS of premenopausal group was 78.674.35mg/dl and of postmenopausal group was 79.476.81 mg/dl

The PPBS of premenopausal was 97.349.56 mg/dl and of postmenopausal group was 97.253.06. mg/dl.

Both the study and control groups were matched with respect to BMI, FBS, PPBS.

Conclusion

In our study there was no significant difference in BMI, FBS, PPBS in both premenopausal and postmenopausal women.

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