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Study of High Sensitive C-Reactive Protein in Cardiac Post-Menopausal Women and Compare to Healthy Post-Menopausal women

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Abstract

Background: Women have a markedly increased risk of cardiovascular disease (CVD) after menopause and often develop coronary heart disease many years later than males. CVD is the primary cause of death in women. The menopausal transition (MT) is thought to be a contributing factor to the elevated risk of coronary heart disease, based on this observation..

Methods: This paper focuses on analyzing the importance of biochemical parameters in post-menopausal heart disease including HS-CRP and their implications in the evolution of the disease by using standard procedure of selected biochemical parameters.

Results: The present study showed that the value of HS-CRP was significantly high in post-menopausal heart disease patient compare to normal post-menopausal patients.

Conclusions: Our study also shows that post-menopausal heart disease patient patient have a high risk of critical condition, heart attack and developing sever disease and also show poor prognosis compared normal post-menopausal patent.

Keywords: HS-CRP, MT, PMHD,CVD

Introduction

It is thought that the proper amount of endogenous estrogen released throughout the menstrual cycle has a cardioprotective impact on premenopausal women. This may be the cause of fertile women's lower incidence of coronary heart disease than that of males [1]. But as a person reaches menopause, their ovaries stop producing a considerable quantity of estrogen, making them more vulnerable to conditions like osteoporosis, dyslipidemia, and heart disease that are linked to low estrogen levels [2, 3]. Following menopause, there are major hormonal changes that impact plasma lipid and lipoprotein metabolism, ultimately leading to cardiac-related diseases. These changes include reduced plasma estrogen levels and higher levels of luteinizing hormone (LH) and follicle stimulating hormone (FSH) [4, 5].

By preserving low levels of TAG and LDL-C and high levels of HDL-C, estrogen has a cardioprotective impact. Increased expression of LDL receptors on cell surfaces and an accelerated conversion of hepatic cholesterol to bile acids are likely the causes of the mass clearance of LDL-C from the plasma. HDLC is made possible by an increase in apolipoprotein A-I synthesis and a decrease in hepatic lipase activity [6]. Estrogen influences lipid and lipoprotein metabolism by causing the apoprotein gene to be expressed in the liver [1].

Following menopause, this cardiac protective function disappears, placing postmenopausal women at increased risk for crippling and frequently fatal cardiovascular disease (CVD) consequences [7]. By directly regulating the mRNA production of certain proteins, such as lipoprotein lipase (LPL) and hormone-sensitive lipase (HSL) in adipose tissue, estrogen hormone modulates the cellular level. By inducing the production of other hormones including glucagon, growth hormone (GH), and catecholamines, which raise HSL activity, estrogen also indirectly affects adipose tissue. The rate at which structural apolipoproteins for VLDL and HDL are synthesized in the liver is controlled by 17-beta-estradiol, a main circulating type of estrogen. It lowers the pace at which apoB-100 is synthesized, which lowers the concentration of VLDL, a risk factor for atherosclerosis.

On the other hand, it accelerates the synthesis of apoA-I and apoA-II, raising HDL content and providing atheroprotective effects. By reversely transporting cholesterol from peripheral tissue to the liver, HDL, which contains apoA-I and apoAII, aids in the breakdown of cholesterol found in VLDL and chylomicron [8].

The primary circulating lipids in our body are TAG and TC, whereas the lipoproteins that carry cholesterol include chylomicron, VLDL, IDL, LDL, and HDL. Total plasma cholesterol is determined by the amount of cholesterol present in VLDL, IDL, LDL, and HDL. Endogenous cholesterol is transported by LDL from the liver to the peripheral tissues. In some cases, LDL causes cholesterol to accumulate in the artery's intimal layer, which starts the atherosclerotic process. Due to its reverse cholesterol transport activity, HDL has the ability to prevent atherosclerosis by removing excess cholesterol from cells, especially cholesterol-engulfed macrophages in atherosclerotic lesions, and transferring it into the liver where it is excreted by the liver through bile acid. The ratio of these two lipoproteins controls the onset, course, and consequences of atherosclerotic plaque [9].

When cholesterol esters build up in the artery's intimal layer, a fatty streak lesion is created, signifying the beginning of atherosclerosis. If the fatty stripe doesn't go away over time, it could eventually turn into atherosclerotic plaque. If the plaque keeps getting bigger, it could obstruct the vessel or cause thrombosis, which could result in more ischemia disease complications such peripheral artery disease, coronary heart disease, or ischemic stroke [9].

In 2012, the estimated 17.5 million deaths worldwide attributed to CVD were caused by this cause. Countries with low and moderate incomes account for more than 75% of deaths from CVD [10]. In the USA, 30% of women acquire osteoporosis and 70% of women develop cardiovascular disease after menopause, according to a 2002 American Heart Association report [3].

CVD is primarily associated with dyslipidemia, particularly hypercholesterolemia. The most common and dangerous health issue, cardiovascular disease affects people everywhere, including Nepal. In Nepal, women make up more than 50% of the population, with a greater proportion of those over 50. Particularly, because they are retired, this population lacks physical activity, and eating unhealthy foods increases their risk of atherogenic effects. Given that this is the average age for menopause, it is likely that more Nepalese women are susceptible to consequences from cardiovascular disease, which is a cause for concern [11].

Since estrogen is essential for the metabolism of fats and lipoproteins, it is critical to keep an eye on the lipid profile in postmenopausal women, who often have lower estrogen levels. In order to compare the serum levels of TC, TAG, HDL-C, and LDL-C in premenopausal and postmenopausal women, the current study was conducted.

Materials And Methods

A study was conducted in Pacific Institute of Medical Sciences, Rajasthan, from March 2019 to December 2021 on Post Menopause with cardiovascular disease patient. The source population was all cases of cardiovascular disease admitted at PIMS with a confirmed diagnosis of cardiac disease reported by central laboratory and cardiology department.

A total number of 200 patients admitted at Pacific Institute of Medical Sciences Udaipur, was form the subjects of the present study. Out of these 100 patients were suffering from heart disease, and 100 were normal patients. Efforts will be made to match

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all anthropometric factors comparable to both the groups of patients.

Group 1: Confirm post-menopausal 50-70 years' healthy women patients.

Group 2: Confirm post-menopausal women with coronary heart disease age 50-70 years.

Inclusion Criteria

1. Patient who are willing to participate.
2. Post-menopausal women with coronary heart disease diagnosed by cardiology department.

Exclusion Criteria

1. Patients below 50 years and above 70 years of age was excluded in the study.
2. Metabolic diseases, malnutrition, or histories of consuming vitamins or minerals supplements, regular steroids were excluded from the study.
3. Cancer patients was excluded.
4. Diabetes mellitus, liver diseases, kidney disease, rheumatoid arthritis, patients were excluded.
5. To minimize the effect of life style on lipid profile trained athletes or sports women was excluded from the study.
6. Post-menopausal women with thyroid dysfunction and those who are taking antihypertensive drug was excluded.

Blood Collection, Separation and Storage of Samples: After obtaining informal consent from all patients and healthy control, 5 ml of venous blood will be collected in a sterile plain bulb under all aseptic precautions. Blood will be drawn from anticubital vein in plain vial. After samples collection, samples will be centrifuge at 3000 RPM for a period of 15 minutes. Serum will be separated after centrifugation.

Clinical Methodology

Serum HS-CRP was recorded by using Autoanalyzer ERBA EM-200

Statistical Analysis

For the quantitative analysis, we used the software SPSS software. In this meta-analysis, all p values reported were two-tailed with the statistical significance set at $\leqslant 0.05$.

Result

The present study showed that the mean value and standard deviation of HS-CRP (3.0±1.9) was significantly high in post menopause cardiac women with P value (< 0.0001) compare to normal women with post menopause (1.2± 0.74). (Showed in Table .1 and Figure.1)

Table 1: Comparison of HS-CRP between Normal Patient and post-menopausal heart disease patients.

S.No	Test	Normal Patient		Pmhd Cases		P Value
		MEAN	SD	MEAN	SD	
1	HS-CRP	1.2	0.74	3.0	1.9	P < 0.0001

Fig 1: Comparison of HS-CRP between Normal Patient and post-menopausal heart disease patients.

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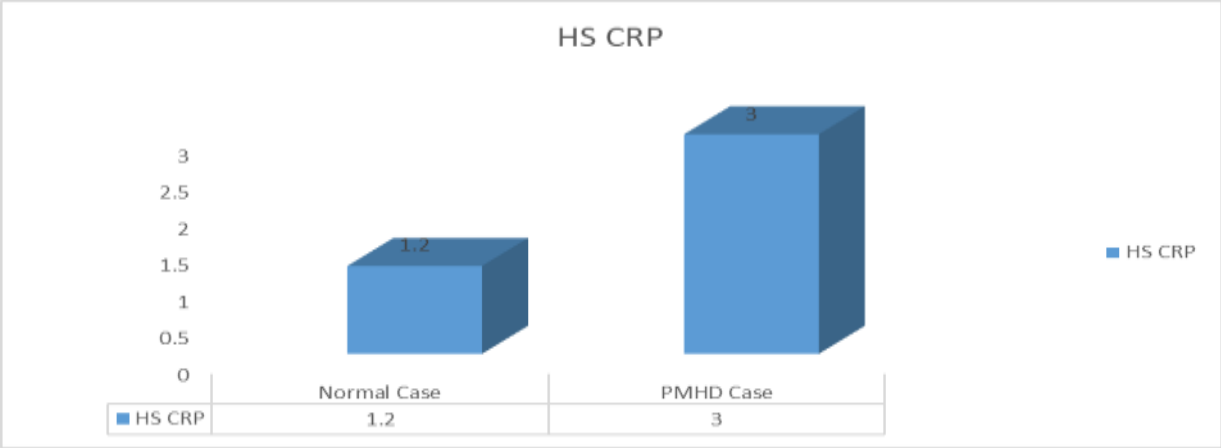

Discussion

The present study showed that the mean value and standard deviation of HS-CRP (3.0±1.9) was significantly high in post menopause cardiac women with P value (< 0.0001) compare to normal women with post menopause (1.2± 0.74).

The current study's findings are consistent with a published study of [12], which found that there were significant differences between pre- and postmenopausal women's levels of the studied hs-crp, with postmenopausal healthy women's serum levels of hs-crp approximately three times higher than pre-menopausal women's..However, this investigation was similar to that of [13], which discovered no appreciable variations between the pre and post.

A high level of HPC in the postpartum state can be attributed to the lifestyle choices that lead to lipid deposition, the lack of estrogen, and the function of lipids in the synthesis of proinflammatory mediators such as HPC. Additionally, the ovaries' diminished function and the notable alterations in sex hormone. CRP attaches itself to oxidized low-density lipoprotein (LDL), increasing adhesion molecules that promote complement proteins and inflaming atherosclerotic plaques. Additionally, CRP stimulates the creation of nitric oxide and increases adhesion molecules. It also promotes the induction of tissue factor, a remarkable factor on the surface of monocytes that is thought to be one of the major coagulation factors.[12]. When the regulating impact of estrogens on hs-crp is eliminated, postmenopausal women's levels of hs-crp rise and their risk of cardiovascular disease (CVD) increases. The cross-sectional design of the current study makes it difficult to assess how hs-crp and menopause are related. A lengthy investigation with a sizable population and sample size is required for it. It is important to note that postmenopausal women have a clearly increased risk of cardiovascular disease (CVD), with significantly higher pre- and post-hs-crp levels than women of other ethnicities in other parts of the world. Rifai et al. & Michelle et al., [14,15] as well as the late suggestion of the American Heart Association (AHA) that those women are less obviously ill and are candidates for CVD prevention, all clearly demonstrated this. It has been demonstrated by Goff et al., [16] that CRP is a significant predictor and one of the independent and significant risk factors for patients to develop CVD (rr=4.4 in the future); LDL is ranked second in this regard, behind hs-crp [17, 18].

Conclusion

The present study done on Normal post-menopausal patient and post menopause heart disease patient admitted in Pacific Institute of Medical Sciences, Umarda, Udaipur. Total 200 patients were included for this study .100 was normal post-menopausal patient and 50 was post menopause heart disease patient. 50-70 age group was taken for this study The present study showed that the mean value and standard deviation of HS-CRP (3.0±1.9) was significantly high in post menopause cardiac women with P value (< 0.0001) compare to normal women with post menopause (1.2± 0.74).

And this study also showed that the post-menopausal women had an increased hazard to die from heart attack after menopause.

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Ethical Issues: Research project approved by the ethics committee of Pacific Institute of Medical Sciences, Umarda Udaipur- 313005, Rajasthan, INDIA**.**

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