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Hepcidin Overexpression and Hypotestosteronemia in Middle-Aged Atherosclerosis Patients

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ABSTRACT

Atherosclerosis is a dangerous condition that can endanger the lives of both men and women. As people age, their risk of developing this illness rises. In order to evaluate the vital markers pretentious by Cardiopathy, such as hepcidine, iron, and sex hormones. Forty men and twenty women with atherosclerosis, ages 45 to 75, took part in the current study. The control group, on the other hand, included fifteen healthy men and fifteen healthy females. The data showed that the patients' hepcidin levels differed considerably from those of the healthy individuals. The study's findings also demonstrated that males with atherosclerosis have low levels of the sex hormone testosterone. Although males with atherosclerosis are more prone to experience sexual dysfunction as they age, the current study found that individuals with atherosclerosis are more vulnerable to oxidative stress because of higher iron levels.

INTRODUCTION

The buildup of fat and cholesterol in the arteries causes atherosclerosis, a severe condition that obstructs the body's ability to receive oxygen-rich blood [1]. According to epidemiological research, women are protected from heart disease by the female hormone estrogen, while men are more likely than women to develop atherosclerosis. Thus, the risk factor for atherosclerosis in postmenopausal women. Arteriosclerosis can be caused by a variety of factors, including smoking, inactivity, poor diet, and high blood pressure [2].

Atherosclerosis is caused by the accumulation of a yellow material on the artery walls, according to a prior study [3]. Further investigation has revealed that this substance is fat [4]. Furthermore, this demonstrated that additional risk factors for atherosclerosis, such as elevated body iron levels. Iron is a crucial nutrient. It can be hazardous even though it is essential for processes including electron transport, oxygen transport, and DNA synthesis [5]. Iron homeostasis is complicated and needs to be managed. Iron metabolism problems may be a significant contributor to human disease. Although iron has been

involved in atherosclerotic lesions for decades, its exact involvement in atherosclerosis and coronary artery disease is still unknown. According to Sullivan, iron is a cardiovascular risk factor. A relative iron deficit may prevent cardiovascular disease, but a modest increase in stored iron may cause it [6]. Hepcidin is a hormone that traps iron in macrophages, reduces the absorption of iron in the intestines, regulates iron homeostasis, and reduces serum iron levels. Thus, by producing hydroxyl radicals through the Fenton reaction, Lipid peroxidation and inflammation may be accelerated by the iron generated by the fleshy tissue [7], [8]. Atherosclerosis has recently been associated with elevated serum hepcidin levels [10].

METHODOLOGY

Subjects

Sixty patients with atherosclerosis from Iraq, 20 women and 40 men, participated in the study. They are between 45 and 75 years old. "The AL-Sader Teaching Hospital in Najaf City, Iraq", identified these patients as having atherosclerosis. On the other hand, thirty people who appeared to be in good health, 15 men and 15 women, were designated as the control healthy group. They share a similar age variety as the first group.

Biochemical measurements

After coagulation, the blood was used to separate the serum. Using kits in the ELISA apparatus, hepcidin and sex hormones were measured [11]. A spectrophotometer was used to estimate the iron content. [12].

Statistical analysis

The mean ± standard deviation was used to express the results. The probability (p) value was computed using SPSS and Microsoft Excell® 2016 software, and the pooled T-Test was utilized to compare the statistical parameters between the patient and control groups. The P-value stayed less than 0.05; the variation deemed significant [13].

RESULTS AND DISCUSSION

Table 1 shows that Hepacidin and testosterone levels in the patients were considerably greater than in the control group. Furthermore, these findings show that patients' levels of iron and estradiol did not significantly differ from those of the control group.

Table 1 Hepcidin, iron, and some sex hormone levels in atherosclerosis patients as compared to the control group

Parameters	Patients(60)	Controls(30)	P Value
	Mean ± SD	Mean ± SD	
Hepcidin ng/mL	281.74 ± 65.22	208.71 ± 84.27	0.0001
Testosterone ng/dL	3.35 ± 2.35	2.27 ± 2.29	0.038
Estradiol pg/mL	12.52 ± 15.22	7.13 ± 12.56	0.079
Iron µg/dL	89.41 ± 47.83	92.27 ± 33.83	0.742

These findings are consistent with prior research. Clara et al. (2020) have demonstrated that elevated iron levels lead to increased hepcidin synthesis and decreased iron release from macrophages and enterocytes [14]. Hepcidin, on the other hand, is essential for iron homeostasis [15]. By attaching to Ferroportin, the sole source of iron released by macrophages, hepcidin controls blood iron levels by inhibiting it and causing iron release into the bloodstream to be suspended. However, when iron storage is limited, hepcidin synthesis is down-regulated and these cells release more iron [16].

Because hepcidin kept iron levels within acceptable ranges, the current investigation demonstrated that hepcidin levels were significantly greater in atherosclerosis patients while iron levels were within normal ranges [17].

Table 2 shows some biomarker levels in female atherosclerosis patients relative to the control healthy group.

Variations	Patients / F	Controls / F	(P-Value)
	"Mean ± SD."	"Mean ± SD."	
Hepcidin ng/mL	260.31 ± 58.18	186.77 ± 87.31	0.009
Testosterone ng/dL	0.63 ± 0.97	0.32 ± 0.22	0.214
Estradiol pg/mL	9.58 ± 11.12	10.74 ± 16.61	0.827
Iron µg/dL	104.74 ± 57.49	96.65 ± 22.73	0.574

The data in Table 2 show a significant increase in hepcidin levels, but there was no appreciable difference in some markers between the female patients and the healthy control group.

Female sex hormones have been shown in earlier research to prevent heart disease in women, and this protection diminishes as a woman gets closer to menopause [18]. The results show that, compared with the control group, women with atherosclerosis do not significantly change their estradiol levels. This is because the study's participants were postmenopausal, with ages ranging from 45 to 75 [19].

Table 3 shows some biomarker levels in male atherosclerosis patients relative to the control healthy group.

Variations	M Patients	M Controls	P Value
	Mean ± SD	Mean ± SD	
Hepcidin ng/mL	294.96± 71.01	197.33 ± 93.82	0.002
Testosterone ng/dL	4.47 ± 1.33	5.52 ± 1.71	0.044
Estradiol pg/mL	11.66 ± 7.32	14.24 ± 6.69	0.224
Iron µg/dL	81.72 ± 40.92	87.81 ± 42.51	0.637

The sex hormone levels in males with atherosclerosis were shown in Table 3. Hepcidin levels significantly increased, testosterone levels significantly decreased, and estradiol levels did not significantly alter when males with atherosclerosis were compared to the control group. Iron levels, however, did not significantly change.

These findings are consistent with those of other earlier research [2, 19, 20]. In addition to other factors like obesity, testosterone is thought to be one of the primary causes of heart disease in men. Because obese men have decreased levels of SHBG-bound testosterone in addition to free and total testosterone, this may be brought on by elevated aromatase activity, which changes testosterone into estrogen. Previous studies have indicated that obese men had a higher risk of heart disease [21]. According to earlier research, men who have low testosterone levels have impotence as they age. [22, 23].

CONCLUSION

The current investigation found that atherosclerosis patients' serum hepcidin levels were significantly higher. Furthermore, it has been shown that men with atherosclerosis deficiency of the male sex hormone (testosterone) It increases their risk of impotence as they become older. Furthermore, because of increased iron levels, Atherosclerosis patients are more vulnerable to oxidative stress; nonetheless, age-related sexual dysfunction is more common in men with atherosclerosis.

CONFLICT OF INTEREST:
The authors declare no conflict of interest.

FUNDING:
The study self-funded.

ETHICAL CLEARANCE:

Permission was taken from the concerned authorities. Patients were also informed about the purpose of the study and they all gave their consent to participate in the study.

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