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Estimation levels of Lipid Profile, LH and FSH in Men with Polycythemia at Maysan Province (Iraq)

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Abstract

Background: There exist two primary classifications of polycythemia: primary and secondary polycythemia. These conditions share the common characteristic of an abnormally elevated concentration of hemoglobin and/or hematocrit within the peripheral blood. It's worth noting that secondary polycythemia is significantly influenced by smoking, a known factor that diminishes oxygen delivery to tissues. Furthermore, smoking's detrimental effects on oxidative stress, blood coagulation, and blood lipid composition amplify the risk of developing atherosclerotic diseases, thereby underscoring the importance of addressing this hazardous habit in the context of overall health and well-being.

Aim: This study aims to illuminate the intricate relationship between smoking, lipid profiles, and reproductive hormones, shedding light on potential health implications and providing valuable data to inform clinical management strategies for individuals facing this condition.

Material and Methods: A research investigation was carried out involving a cohort of 100 male participants, comprising 75 individuals diagnosed with polycythemia and 25 control subjects. The age range of the participants ranged from 20 to 59 years. Blood samples were procured from the blood bank in Maysan Province (in Iraq) and gathered during the period extending from November 2022 to March 2023.

Results: The result showed that total cholesterol, triglyceride, low-density lipoprotein and very low-density lipoprotein increased significantly ($p < 0.05$). While high-density lipoprotein decreased significantly ($p < 0.05$) in both groups of polycythemia (smokers and non-smokers) compared with the control group. No significant ($p > 0.05$) variation was recorded in the values of follicle-stimulating hormone, testosterone and estrogen in the smoker and non-smoker groups compared with the control group. The levels of luteinizing hormone in smoker and non-smoker groups were increased significantly ($p < 0.05$) in comparison to the control group.

Conclusion: Our investigation has revealed notable alterations in the lipid profiles of men affected by polycythemia, regardless of their smoking status. However, when it comes to changes in reproductive hormones within the studied population, these adjustments did not attain statistically significant levels, except for luteinizing hormone, which exhibited a noteworthy variation.

Keywords: Smoking, Lipid, Polycythemia, Reproductive, Hormon

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1. Introduction

Polycythemia, characterized by elevated levels of hematocrit and/or hemoglobin in peripheral blood, is commonly categorized as either primary or secondary [1]. Secondary polycythemia occurs as a result of increased production of erythropoietin, typically triggered by tissue hypoxia originating from various factors such as cardiovascular conditions, high-altitude environments, smoking, and sleep apnea [2]. Smoking is a prominent contributor to secondary polycythemia due to the tissue hypoxia it induces [3,4].

Smoking, in particular, is a significant factor in the development of secondary polycythemia. Smokers experience reduced oxygen transport in the bloodstream and diminished release of oxygen to tissues when compared to non-smokers. Moreover, smoking heightens the risk of atherosclerotic diseases through multiple mechanisms, including oxidative stress, increased blood coagulation tendencies, and detrimental alterations in the blood lipid profile [5,6]. Previous research has indicated that smoking is closely associated with dyslipidemia, a condition characterized by elevated levels of low-density lipoproteins (LDL), triglycerides, and reduced levels of high-density lipoproteins (HDL). In essence, the impact of smoking extends beyond its link to secondary polycythemia; it significantly contributes to the development of atherosclerosis by promoting dyslipidemia and other vascular abnormalities, thereby emphasizing the critical need for smoking cessation to reduce cardiovascular risks [7].

Testosterone, the primary male androgen hormone, is produced by Leydig cells in the testes. Its production is regulated by hypothalamic gonadotropin-releasing hormone (GnRH), which, in turn, controls the release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the anterior pituitary gland. This hormonal system, known as the hypothalamic-pituitary-gonadal (HPG) axis, is vital for maintaining male sexual development and function. GnRH signals the pituitary to release FSH, supporting sperm production, and LH, which stimulates Leydig cells to produce testosterone. This testosterone plays a crucial role in male sexual characteristics, libido, and overall sexual health. Understanding the HPG axis is fundamental to comprehending male reproductive biology and health [8]. Testosterone plays a crucial role in the production of red blood cells in males, and it's worth noting that polycythemia, an abnormal increase in red blood cell count, is a known unintended consequence associated with testosterone therapy for hypogonadism [9,10]. Moreover, elevated hematocrit levels and increased serum testosterone are linked to a condition known as persistent mountain illness [11].

It's important to recognize that the stress induced by hypoxia (low oxygen levels) can impact various stages of the reproductive process. This influence extends to areas such as gametogenesis, the ovarian cycle, menstruation, birth weights, stillbirth rates, infant mortality, postpartum behavior, menopause, and gonadal hormone regulation. These effects are mediated by key transcription factors known as hypoxia-inducible factors (HIFs), which orchestrate changes in gene regulation in response to hypoxic stress during both adult and embryonic development. Understanding these connections between hypoxia and reproductive processes sheds light on the intricate interplay between oxygen levels and reproductive health [12].

2. Materials and Methods

Subjects

This study took place at the Maysan Blood Bank located at Maysan Province/ south of Iraq and involved 100 male participants aged 20-59. Out of these, 75 had polycythemia, while 25 were healthy controls. The study spanned from November 2022 to March 2023, aiming to investigate differences in blood parameters between the two groups and provide insights into polycythemia within this demographic.

Permission to conduct this study was issued by the health institutional Center for Blood bank in Maysan Province in order to facilitate mission number 2450 on 3.11.2022.

Blood sample

A blood sample of approximately 5-6 milliliters was collected from each participant and then transferred into a gel tube. Following this, the samples were subjected to centrifugation, and the resulting serum was utilized for the analysis of both lipid profile and hormones. For lipid profile assessment, in vitro tests, were conducted to quantitatively measure total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and high-density lipoprotein (HDL) concentrations in the serum. These measurements were performed using photometric systems, as outlined in references [13] and [14].

In parallel, the analysis of hormones including follicle-stimulating hormone (FSH), luteinizing hormone (LH), testosterone, and estrogen was carried out. This hormone quantification was accomplished using a Chemiluminescent Immunoassay Analyzer (CLIA), providing precise measurements of hormone levels in the serum, as described in reference [15].

Statistical Analysis

The data was analyzed statistically to know the significance of the different parameters by one way ANOVA, the difference were considered to be significant at $P < 0.05$ the values present as means \pm SE .

3. Results

In the current study, the result showed that TC, TG, LDL and VLDL which increased significantly ($p < 0.05$). While HDL decreased significantly ($p < 0.05$) in both groups of polycythemia (smokers and non-smokers) compared with the control group, as shown in Table (1).

Table 1: The values of Lipid profile in male with polycythemia and control groups.

Parameters	TC (mg/dl)	TG (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Groups					
Control	b 148.09 \pm 3.28	b 129.76 \pm 7.83	a 44.49 \pm 0.75	b 101.01 \pm 3.33	b 25.95 \pm 1.57
Polycythemia Smokers	a 175.50 \pm 4.07	a 182.27 \pm 11.93	b 38.54 \pm 1.29	a 119.26 \pm 3.91	a 36.45 \pm 2.39
Polycythemia Non Smokers	a 185.81 \pm	a 201.15 \pm	b 37.96 \pm 1.58	a 130.03 \pm	a 40.23 \pm 4.45

	6.93	22.25		5.70	
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Means \pm SE

The different letters refer to significant difference among group at level of ($p < 0.05$)
The similar letters refer to non-significant difference among group at level of ($p < 0.05$).

Hormonal parameters

The results revealed that there were no significant ($p > 0.05$) variation in the values of FSH in the smoker and non-smoker groups (5.32 ± 0.39 , 6.53 ± 0.82 mlU/ml, respectively) in comparison with the control group (6.18 ± 0.54 mlU/ml) as shown in **Fig 1**.

In addition, no significant ($p > 0.05$) variation was recorded in the values of testosterone in the smoker and non-smoker groups (2.65 ± 0.15 , 2.72 ± 0.23 pg/ml, respectively) in comparison with the control group (2.45 ± 0.16 pg/ml) as illustrated in **Fig 2**.

No significant ($p > 0.05$) variation was recorded in the values of estrogen in the smoker and non-smoker (45.76 ± 1.47 , 44.76 ± 2.36 pg/ml, respectively) in comparison with the control group (43.40 ± 2.17 pg/ml) (**Fig 3**). The levels of LH in the smoker (7.63 ± 0.41 mlU/ml) and non-smoker groups (8.95 ± 0.92 mlU/ml) were increased significantly ($p < 0.05$) in comparison with control group (5.01 ± 0.24 mlU/ml). while the smoker and non-smoker groups did not differ significantly ($p > 0.05$) between them, (**Fig 4**).

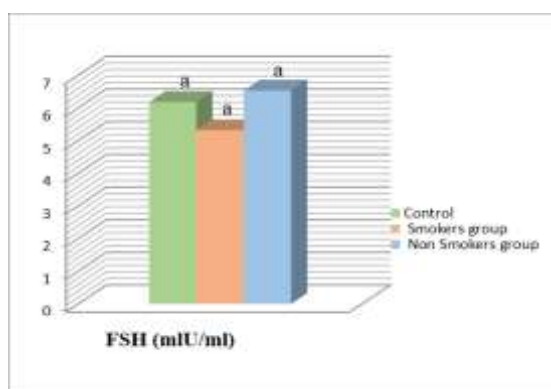


Fig (1):The levels of Follicle stimulating hormone (FSH) in men with polycythemia and control.

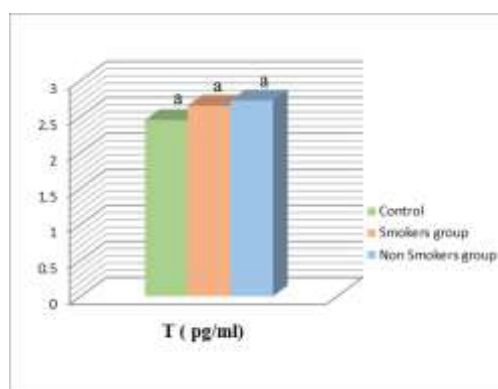


Fig (2): The levels of testosterone(T) in men with polycythemia and control.

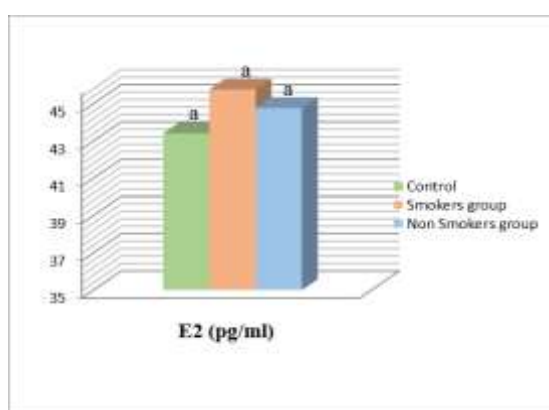


Fig (3):The level of Estradiol (E2) hormone in men with polycythemia and control.

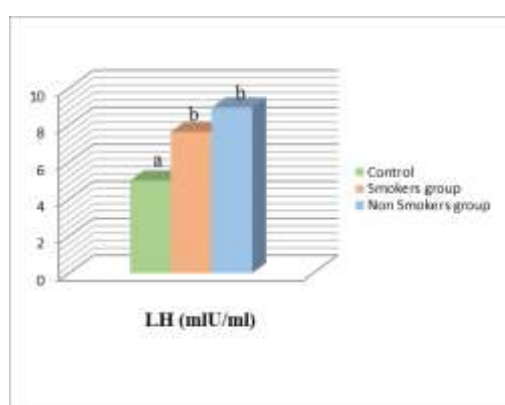


Fig (4): The level of Luteinizing hormone (LH) in men polycythemia and control.

4. Discussion

The current study findings align with prior research that observed significantly lower levels of HDL in heavy smokers with polycythemia [16]. These results are consistent with other studies as well [17,18]. Smoking has been established to elevate levels of total cholesterol (TC), triglycerides (TG), and low-density lipoprotein (LDL) in smokers while simultaneously reducing serum HDL levels when compared to non-smokers. One plausible explanation for these effects is that smoking induces alterations in lipid levels through the activation of multiple pathways. Among these, the primary pharmacologically active component in cigarettes, nicotine, stimulates the sympathetic nerves of the adrenal glands, triggering the release of hormones such as epinephrine and norepinephrine. This, in turn, increases the activity of lipase enzymes and the concentration of free fatty acids in the bloodstream, ultimately leading to elevated levels of triglycerides and lipids while concurrently decreasing the levels of beneficial high-density lipoprotein (HDL)[19].

The comparison of FSH levels between male smokers and non-smokers did not reveal a significant difference when compared to the control group. These findings align with a study conducted by [20], which similarly reported no correlation between smoking and FSH levels in a cross-sectional analysis involving 255 men. However, an interesting observation from a previous study indicated that individuals who smoked more than 10 cigarettes per day exhibited a 37% reduction in FSH levels compared to those who consumed fewer than 10 cigarettes per day [21]. In contrast, contrary results were reported in another study, which found that nonsmokers had lower FSH levels compared to smokers[22].

As for testosterone levels, our results are consistent with the study conducted by Harman and his research group [23]. In their population-based investigation involving a total of 890 men, smoking did not appear to have a substantial impact on testosterone levels. Additionally, another study cited [24] found no association between tobacco use and reduced testosterone levels in their investigation. Higher testosterone levels were observed in current and former smokers when data from 1,104 men were analyzed [25]. More than 1,000 males were seen in a clinic for sexual dysfunction, and those who smoked had higher testosterone levels [26]. Our findings were similar to another study by [27], Serum levels of estrogen were not significantly different between smokers and nonsmokers.

The result of LH in this study agrees with a study performed by [28] that revealed an elevation in the level of LH hormone in smoking compared to control. The increase in LH concentration is consistent with a previous study [29]. But, contrary to the study done by [20], they found no significant effect of smoking on LH levels in adult men.

According to [30], the higher levels of LH observed in smokers may be due to testicular dysfunction. Increased levels of LH in men typically indicate a deficiency in negative feedback from male steroid hormones [31]. Smoking was thought to promote LH secretion by activating Leydig cells [25]. Nicotine in cigarettes may also increase dopamine release by activating nicotinic acetylcholine receptors on neurons in the mesolimbic pathway. This may influence gonadotropin (LH) secretion and modify the hypothalamic-hypophysial-gonad feedback pathway, hence influencing testosterone synthesis [32].

5. Conclusion

In the present study, a noteworthy association between polycythemia and lipid profiles was observed, with a particular emphasis on individuals who smoke. The findings indicated a

substantial increase in the levels of total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and very-low-density lipoprotein cholesterol (VLDL), accompanied by a concurrent decrease in high-density lipoprotein cholesterol (HDL-C). These lipid alterations shed light on the intricate relationship between polycythemia and lipid metabolism, particularly among smokers.

Additionally, our study revealed an elevation in luteinizing hormone (LH) levels, suggesting a potential hormonal response in individuals with polycythemia. Intriguingly, this hormonal shift did not significantly impact the levels of follicle-stimulating hormone (FSH), total testosterone (TT), or estradiol (E2). These findings highlight the multifaceted nature of the hormonal changes associated with polycythemia and underscore the importance of considering smoking status in the assessment of these metabolic and hormonal variations. This research contributes to our understanding of the intricate interplay between polycythemia, lipid profiles, and hormonal dynamics, particularly in the context of tobacco use.

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