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LIPID PROFILE IN EGYPTIAN PATIENTS WITH PROSTATE CANCER AND BENIGN PROSTATE HYPERPLASIA

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ABSTRACT

Prostate cancer incidence is thought to be associated with western diet, which is rich with saturated fat. There is a possible relation between high cholesterol and prostate cancer androgens that play a role in prostate tissue and cancer are synthesised from cholesterol. Hypercholesterolemia may possibly alter prostate morphology by affecting the sex steroids axis and thus contributes to benign prostate hyperplasia**.** Moreover, cholesterol is synthesized by tumour cells but also enters cells from the circulation.

Due to changes in Egyptians dietary habits and life style there is a great need to evaluate changes in serum lipids in prostate cancer patients. In this study, we measured serum cholesterol, triglycerides, HDL-C and LDL-C in patients with benign prostate hyperplasia, androgen dependent prostate cancer and androgen independent prostate cancer to evaluate these changes.

Results indicated that serum cholesterol showed statistically significant increase in androgen dependent group (P = 0.006) and androgen independent group (P = 0.001) as compared to hyperplasia group, while, no significant variation was found between androgen dependent and androgen independent groups (P = 0.246). Serum triglycerides showed significant increase in androgen independent group as compared to hyperplasia group (P = 0.032), while, there is no significant variation was found between androgen dependent and

androgen independent group (P = 0.360) and between androgen dependent group and hyperplasia group (P = 0.211). Serum HDL-C showed statistically significant increase in androgen dependent (P = 0.011) and androgen independent groups (P = 0.001) as compared to hyperplasia group, while, no significant variation was found between androgen dependent and androgen independent groups (P = 0.126). Serum LDL-C showed no statistically significant variation between androgen dependent group as compared with hyperplasia (P = 0.076) and between androgen dependent group as compared to androgen independent group (P = 0.614) while, there was a significant increase in androgen independent group as compared to hyperplasia groups (P = 0.05).

It can be concluded that cholesterol and other serum lipid contents may play a significant role in developing of prostate cancer among Egyptian patients.

Key words: Key words: Prostate cancer, lipids, cholesterol, triglycerides, HDL-C, LDL-C

INTRODUCTION

Fatty food has been the focus of dietary studies of prostate cancer more than any other dietary component. Epidemiologic studies of this relationship were surprisingly consistent and suggested a possible causal association (Committee on Diet, Nutrition and Cancer, 1982; Committee on Diet and Health, 1989).

Since the 1980s, reports have linked increased risk of aggressive Prostate cancer (PCa) to the consumption of animal products and/or fatty food. Fleshner et al., (2004) critically appraised 33 published case–control and cohort studies examining the possible relationship between dietary fat and PCa. Previous studies suggested a statistically significant association between intakes of fatty foods and PCa risk, with several demonstrating a significant association for dairy products and meat (Fleshner et al., 2004). The basis for the apparent association between fat intake and increased PCa risk is unknown, but it could be related to a variety of factors, including circulating cholesterol or androgen levels, intake of fat-soluble pesticides, and/or increased production of reactive oxygen species (Harvei, 1997; Fleshner and Kucuk, 2001; Fleshner et al., 2004). Although the evidence remains

inconclusive, the cumulative weight of these studies suggests the existence of a link between dietary fat and PCa progression.

Coffey (2001) mentioned that prostate cancer incidence has been linked to a western diet, which includes high level of saturated fat. There is a possible relation between high cholesterol and prostate cancer. androgens that play a role in prostate tissue and cancer are synthesised from cholesterol. Hypercholesterolemia may possibly alter prostate morphology by affecting the sex steroids axis and thus contributes to benign prostate hyperplasia (Mitropoulos et al., 2004). Moreover, cholesterol is synthesized by tumor cells but also enters cells from the circulation by low density lipoprotein-receptors mediated endocytosis (Li et al., 2001).

Cells in the prostate, as in other tissues, synthesize cholesterol endogenously via the mevalonate pathway. Synthesis within the body starts with one molecule of acetyl CoA and one molecule of acetoacetyl-CoA,which are dehydrated to form 3-hydroxy-3-methylglutaryl CoA (HMG-CoA). This molecule is then reduced to mevalonate by the enzyme HMG-CoA reductase. This step is the regulated, rate-limiting and irreversible step in cholesterol synthesis.

Cellular cholesterol also derives from absorption from circulating lipoproteins (Simons and Vaz, 2004). Consequently, control of cellular cholesterol content is a balance between metabolic processes intrinsic to the cell and the regulation of cholesterol distribution by the organism. Cholesterol content of cell membranes is determined by a complex set of processes, including synthetic pathways in the endoplasmic reticulum, transfer from lipoproteins to the exoplasmic leaflet, receptor-mediated internalization, intracellular transport mechanisms, and efflux from the cell via secretion of lipoprotein complexes. Extensive evidence indicates that this complex homeostatic mechanism breaks down in aging tissues and in cancer and accumulation of cholesterol has been described in a variety of tumour types (Rudling and Collins, 1996; Dessi et al., 1992; Dessi et al., 1994; and Kolanjiappan, et al., 2003). Cholesterol content has also been reported to be altered in normal tissue surrounding malignancies (Nygren et al., 1997). Early reports of histological observations described substantial (two-fold) increases in cholesterol content in benign prostatic hyperplasia compared with normal prostate (Swyer, 1942). These studies concluded that a relationship existed between cholesterol accumulation in tissues and cellular hyperplasia. Studies of

human and animal tissues have also described increases in cholesterol content of prostatic secretions that correlate with disease, age, or the presence of malignancy (Schaffner and Gordon, 1968 and Dessi et al., 1992). Several studies have reported statistically significant positive correlations between cholesterol intake and cancer risk (De Stefani et al., 1997; Horn-Ross et al., 1997 and Jarvinen et al., 1997), implying that prolonged consumption of cholesterol-rich foods might promote cancer in selected tissues.

The aim of this study is to measure the changes in serum cholesterol, triglycerides, HDL-C and LDL-C in Egyptian patients with benign prostate hyperplasia, androgen dependent prostate cancer and androgen independent prostate cancer to evaluate the role of lipids in prostate cancer.

PATIENTS AND METHODS

Seventy six (76) male urologic patients (age range 53 -80 years, mean 66.25 ± 0.74 years with no significant differences between groups) were involved in this study. Fasting blood sample at morning were collected from Ain Shams university hospitals, as well as Electricity hospital and Cleopatra hospital, Cairo, Egypt. The study included three groups: Group 1: Comprised 30 patients having benign prostate hyperplasia. Group 2: Comprised 31 patients having localized prostate carcinoma Group 3: Comprised 15 patients having androgen-independent prostate carcinoma. All the samples by approval from the committee of ethics of National Research Center, Cairo, Egypt.

Serum cholesterol was assayed by enzymatic, colorimetric method (CHOD/PAP) with cholesterol esterase, cholesterol oxidase and 4-aminoantipyrine. kit purchased from Vitroscient company, Egypt.

Serum triglycerides were assayed by enzymatic colorimetric method (GPO/PAP) with glycerol phosphate oxidase and 4-aminoantipyrine. Liquid stable single reagent. kit purchased from Vitroscient company, Egypt.

High density lipoprotein (HDL) cholesterol was determined by HDL-precipitating reagent kit purchased from Vitroscient company, Egypt. The Vitro HDL-precipitating method utilizes the well established precipitating properties of phosphotungstic acid to precipitate non HDL-cholesterol. This precipitation technique is the

most frequently used method for HDL procedures. The remaining cholesterol in the supernatant, HDL-cholesterol, can then be measured using cholesterol reagent kit.

LDL cholesterol was calculated using Friedewald formula.

LDL-cholesterol (mg/dl)= Total cholesterol – HDL-cholesterol – (triglycerides / 5).

This formula is valid for triglycerides concentrations up to 400 mg/dl.

Statistical analysis

Statistical analysis was performed using the SPSS software package for Windows [SPSS (UK) Ltd., Surrey, United Kingdom]. Analysis was carried out using a t-test for comparing two variables. P value considered significant when it was < 0.05.

RESULTS AND DISCUSSION

Results

Evaluation of lipid profile:

The lipid profile parameters including (cholesterol, triglycerides, HDL-C, LDL-C) of different groups are illustrated in figures: 1-4.

Figures (1) shows the mean value ± SE of serum cholesterol in hyperplasia, androgen dependent and androgen independent groups were (161.93 ± 5.6, 186.8 ± 6.59 and 199.6 ± 8.52 mg/dl) respectively.

Serum cholesterol showed statistically significant increase in androgen dependent group (P = 0.006) and androgen independent group (P = 0.001) as compared to hyperplasia group, while, no significant variation was found between androgen dependent and androgen independent groups (P = 0.246).

Figures (2) shows the mean value ± SE of serum triglycerides in hyperplasia, androgen dependent and androgen independent groups were (114.13 ±_8.75, 129.29 ± 8.19 and 139.46 ± 7.33 mg/dl) respectively.

Serum triglycerides showed significant increase in androgen independent group as compared to hyperplasia group (P = 0.032), while, there is no significant variation was found between androgen dependent and androgen independent group (P = 0.360) and between androgen dependent group and hyperplasia group (P = 0.211).

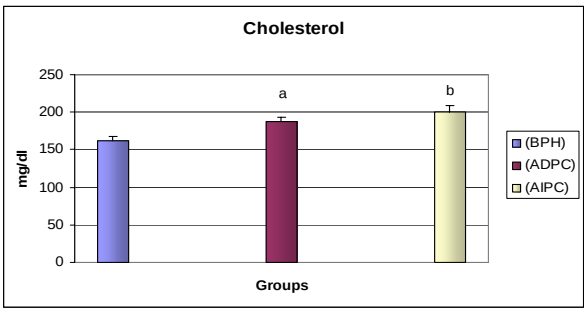

Fig 1): Mean values ± SE of cholesterol in the different groups.
a, significant as compared to hyperplasia group.
b, significant as compared to hyperplasia group.

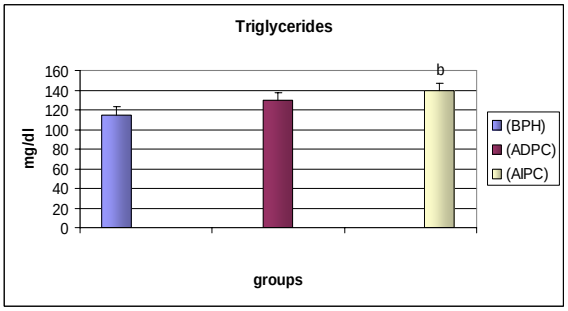

Fig (2): Mean values ± SE of triglycerides in the different groups.
b, significant in compared to hyperplasia group

Figures (3) illustrates the mean value ± SE of serum HDL-C in hyperplasia, androgen dependent and androgen independent groups were (28.9 ±1.22, 35.48 ± 2.15, 41.13 ± 2.87 mg/dl) respectively.

Serum HDL-C showed statistically significant increase in androgen dependent (P = 0.011) and androgen independent groups (P = 0.001) as compared to hyperplasia group, while, no significant variation was found between androgen dependent and androgen independent groups (P = 0.126).

Figures (4) reveals the mean value ± SE of serum HDL-C in hyperplasia, androgen dependent and androgen independent groups were (110.26 ± 5.02, 124.12 ± 5.78 and 129.0 ±7.62 mg/dl) respectively.

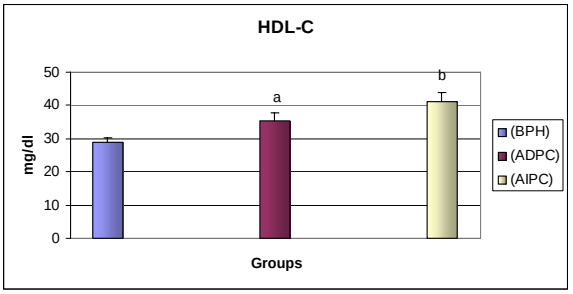

Fig (3): Mean values ± SE of HDL-C in the different groups.
a, significant as compared to hyperplasia group.
b, significant as compared to hyperplasia group.

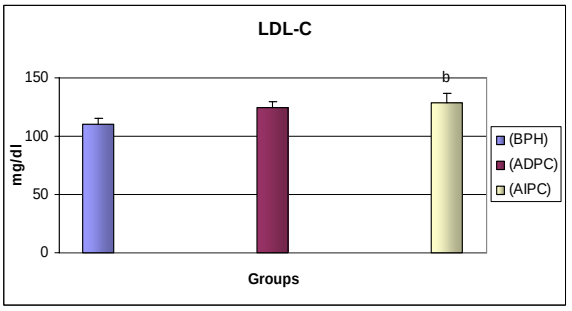

Fig (4): Mean values ± SE of LDL-C in the different groups.
b, significant in compared to hyperplasia group

Serum LDL-C showed no statistically significant variation between androgen dependent group as compared with hyperplasia (P = 0.076) and between androgen dependent group as compared to androgen independent group (P = 0.614) while, there was a significant increase in androgen independent group as compared to hyperplasia groups (P = 0.05).

Discussion

Prostate cancer is the most common malignancy among U.S. men, a 192,280 new cases was detected in 2009 which represented 25% of all new cancer cases (Jemal et al., 2009). In Egypt the GLOBOCAN 2002 database (compiled by Ferlay et al. for the International Agency for Research on Cancer) estimated the number of new cases per year to be 867 cases (www.emro.who.int/ncd/cancer_globocan.htm).

In the period 2002-03 the Egyptian National Cancer Institute at Cairo University reported seeing 238 new cases of prostate cancer out of a total of 9,340 new cancer cases in males (2.6 %). (www.nci.cu.eg)

Chances of developing cancer in men and women have a 35% chance with increasing of age (American Cancer Society 2008). In Egypt aged population represent about 6% of over all population (age 60 years and more) in 2006, it is expected that this percent will be about 9% and 15% in 2015 and 2030 respectively. Between 1986 and 2006 the number of aged populations increased form 2.7 millions in 1986 to 4.4 millions in 2006 (www.idsc.gov.eg/Documents/studies). In spite of the low incidence of prostate cancer in Egypt the increase of the aging population, makes that prostate cancer will become ever more an enormous challenge.

Many studies indicated that, there are associations between lipids and prostate cancer, Dennis et al., (2004) conducted a detailed retrospective review of 29 epidemiological studies investigating the relationship between prostate cancer and fat intake and identified associations of advanced prostate cancer with intake of total and saturated fat. This is supported by the World Cancer Research Fund inquiry (2007) which also reported that a key factor in the development of cancer is total body fat.

Cholesterol is a neutral lipid that accumulates in liquid-ordered, detergent-resistant membrane domains called lipid rafts. A number of studies, going back many years, demonstrate that cholesterol accumulates in solid tumours and that cholesterol homeostasis breaks down in the prostate with aging and with the transition to the malignant state (Martin et al., 2006).

In the present study the level of serum cholesterol revealed statistically significant increase in both androgen dependent group ($P = 0.006$) and androgen independent group ($P = 0.001$) as compared to hyperplasia group, while, there was insignificant variation was found between androgen dependent and androgen independent groups ($P = 0.246$).

In agreement with our results elevated cholesterol levels was a consistent finding in previous studies of men with prostate cancer (Shimano et al., 1996; Swinnen et al., 1997 and Pizer et al., 2001).

Elevated cholesterol levels in prostate cancer cells have been found to result from aberrant regulation of cholesterol metabolism. Subsequent studies of prostate tissues also found increases in

cholesterol content that correlated with the presence of malignancy (Schaffner, 1981). Further studies identified abnormalities in lipid homeostasis as the underlying cause of cholesterol accumulation in the prostate, resulting in massive accumulation of cholesteryl esters (CEs) and a marked increase in free cholesterol levels (Swinnen et al., 1996).

The significance of elevated cholesterol levels in prostate cancer is not fully understood. Increased steady-state cholesterol levels may reflect the high demand for membrane biosynthesis of proliferating cells; however, this is unlikely to be the case in prostate cancer because of the low mitotic index characteristic of this malignancy (Martin et al., 2006).

Is there a reason to believe that cholesterol accumulation, up regulation of cholesterol synthesis or loss of lipid homeostasis is functionally relevant in the promotion of malignant growth? In agreement with the findings from two recent prospective studies it can be suggested that cholesterol may play a promotional role in prostate cancer development and progression. (Platz et al., 2008 and Mondul et al., 2010).

From our results it has been found that serum HDL-C showed statistically significant increase in androgen dependent ($P = 0.011$) and androgen independent ($P = 0.001$) groups as compared to hyperplasia group but, there was insignificant ($P = 0.126$) variation between androgen dependent and androgen independent groups. Serum LDL-C showed no statistically significant ($P = 0.076$) variation between androgen dependent group and hyperplasia group, while androgen independent group shows significant increase in serum LDL-C as compared to hyperplasia group ($P = 0.05$). The process by which internalization of HDL-derived cholesteryl esters (CEs) occurs is known as “selective” cholesterol uptake (Glass et al., 1983; Azhar et al., 2003) and differs from the classic endocytic, LDL receptor pathway in that circulating lipoproteins contribute their (CEs) to cells without internalization of intact lipoprotein particle (Krieger, 1999; Williams et al., 1999; Azhar and Reaven, 2002; Azhar et al., 2003 and Rhainds and Brissette, 2004). Thus, in the selective cholesterol uptake process, lipoprotein cholesterol enters cells unaccompanied by apolipoproteins (Glass et al., 1983 and Azhar et al., 2003).

At least in rodents, selective uptake of HDL cholesterol also plays an important role in the transport of cholesterol to steroidogenic

tissues adrenal gland, ovary and testes providing cholesterol as substrate for steroid hormone synthesis and for storage in cytoplasmic cholesteryl ester droplets (Rigotti et al., 2003). In the rodent adrenal gland, selective uptake accounts for 90% or more of the cholesterol destined for steroid hormone production (Reaven et al., 2000). Considerable evidence now indicates that the conversion of HDL-derived cholesterol into androgens is subject to hormonal (LH/hCG) regulation through the intermediary role of cyclic adenosine monophosphate (Cooke, 1999; Menon et al., 2004).

Indeed, from our study one possible limitation is that circulating total cholesterol concentration may not accurately reflect cholesterol concentrations in the prostate. Although an association between circulating cholesterol concentration and prostate cancer has now been observed, it seems plausible that circulating cholesterol concentration may be correlated with prostate biology. It is important to note that while some authors reported that men with low cholesterol might be less likely to develop prostate cancer, they were unable to assess directly whether lowering of cholesterol might reduce the risk of prostate cancer or not (Mondul et al., 2010).

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مؤشرات الدهون فى مصل دم المرضى المصريين المصابين بالأورام الحميدة و الخبيثة للبروستاتا

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يعتقد أن سرطان البروستاتا من الأمراض المرتبطة بنوعية الغذاء المنتشرة بالمجتمعات الغربية والتي تحتوى على كميات كبيرة من الدهون المشبعة.العديد من الدراسات وجدت أن هناك علاقة محتملة بين سرطان البروستاتا و ارتفاع مستوى الكوليستيرول في الدم. ومن المعروف أن الهرمونات الذكرية - التي تلعب دورا هاما في نمو أنسجة البروستاتا والحفاظ على القيام بوظائفها الحيوية- تصنع من الكوليستيرول، وبالإضافة إلى أن الكوليستيرول يدخل إلى الخلايا السرطانية عن طريق الدم فان هذه الخلايا تقوم بتصنيع الكوليستيرول بنفسها.

نظرا للتغيرات التي حدثت في العادات الغذائية ونمط الحياة بالنسبة للمصريين فإن هناك حاجة ماسة لدراسة التغيرات في مستوى الدهون بالدم في المرضى المصريين المصابين بسرطان البروستاتا.

وجد في هذه الدراسة أن هناك زيادة معنوية ملحوظة في مستوى الكوليستيرول الكلى في مصل الدم في مجموعة سرطان البروستاتا المعتمد والغير معتمد على هرمون الذكورة عند مقارنتها بمجموعة التضخم الحميد للبروستاتا. كما وجد أن هناك زيادة ملحوظة في مستوى الدهون الثلاثية في مصل الدم في مجموعة سرطان البروستاتا الغير معتمد على هرمون الذكورة عند مقارنتها بمجموعة التضخم الحميد للبروستاتا.

كانت هناك زيادة معنوية ملحوظة في مستوى الكوليستيرول المرتفع الكثافة في مصل الدم في كلا من مجموعتي سرطان البروستاتا المعتمد والغير معتمد على هرمون الذكورة عند مقارنتهما بمجموعة التضخم الحميد للبروستاتا.

بالإضافة إلى ذلك كان هناك زيادة ملحوظة في مستوى الكوليستيرول المنخفض الكثافة في مصل الدم في مجموعة سرطان البروستاتا الغير معتمد على هرمون الذكورة عند مقارنتها بمجموعة التضخم الحميد للبروستاتا، بينما لم يكن هناك تغيرات ذات دلالة إحصائية بين مجموعتي سرطان البروستاتا المعتمد والغير معتمد على هرمون الذكورة.

لذا أمكن من خلال هذه الدراسة استنتاج أن مستوى الكوليستيرول ومؤشرات الدهون في مصل دم المرضى المصريين قد تلعب دورا مؤثرا في حدوث سرطان البروستاتا.