

COMPARISON OF THE BLOOD LIPID PROFILE IN CANCER PATIENTS AND HEALTHY PEOPLE.

Pramit Kumar¹ Gunjan Kumar^{1,2}, Pradeep Kumar³ Preeti Sharma^{1,*}

¹ Department of Biochemistry, Santosh Deemed to be University, Ghaziabad, Delhi NCR, India
² Department of Biochemistry, Sri Krishna Medical College, Muzafferpur, Bihar India
³ Department of Biochemistry, Autonomous State Medical College, Fathepur, UP, India
* Corresponding authors.

E-mail addresses: prcdri2003@yahoo.co.in (P. Sharma)

ABSTRACT:

Aim and Objective:

1) To compare the blood lipid profile between cancer patients and healthy individuals.

2) To determine if the lipid profile of the serum changes throughout the pathogenesis of cancer.

3) These changed blood lipid levels can be employed as biochemical indicators of cancer situations.

Introduction:

As important constituents of cell membrane, lipids are crucial for maintaining the integrity of the cell. Alterations in lipid profiles have been associated to cancer because malignancy causes newly multiplying tumor cells to need more lipids for their synthesis, which depletes the body's current resources. It has been demonstrated that cancer patients exhibit hypocholesterolemia. Consequently, the reduced plasma lipid status may serve as a valuable marker to identify the early alterations in the neoplastic process.

KEYWORDS: Serum lipid profile, Cancer, Lipids

Introduction:

With more than 10 million new cases and more than 6 million fatalities worldwide each year, among the leading causes of sickness and death in today's world is cancer. [1]. These fatalities are especially terrible because, in the majority of cases, they could have been avoided with prompt diagnosis and care [2]. In terms of cancer incidence, morbidity, and mortality, lung cancer has historically been rated first, followed by breast cancer, with oral cancer coming in at number six globally [3-5]. Proliferation, apoptosis, and differentiation are all intricate processes that play a role in the complex mechanism that determines how a cancer develops and progresses [6].

Metabolic reprogramming is one of the primary characteristics of cancer, and it can be caused by a variety of oncogenic mutations that result in the abnormal activation of several signalling pathways. To fulfil the demands of vigorous cell growth, cancer cells must rewire their metabolism to produce enough ATP and intermediates for macromolecular production. In addition to causing tumor progression, changes in cellular metabolism also support aggressive characteristics such as invasion, metastasis, and cancer cell resistance to therapeutics [2-4]. Additionally, based on their

surroundings, cancer cells might adopt a variety of metabolic strategies. This adaptability enables cancer cells to flourish in a challenging environment, such as one with oxidative damage or exposure to drugs. The interesting thing about metabolic adaptability is that it is a way for cancer cells to endure in the presence of anticancer medications before regrowing as a result of acquiring new mutations [7-12].

The newly multiplying tumor cells would require a number of fundamental elements much over the range normally utilized in physiological processes. Lipids are one such element [13]. For a number of biological activities, involving cell division and proliferation in both healthy and malignant tissues, lipids are crucial components of cell membranes [14]. Because more lipids are required to satisfy the requirements of these more cells, it would be predicted that the current lipid reserves would become smaller [13].

Studies suggest that increasing dietary fat or cholesterol may increase the chance of developing breast cancer, prostate cancer, or colon cancer. Low total cholesterol (TC) or triglyceride (TG) levels and cancer morbidity have been positively correlated in earlier studies [15-22]. For example, excessive cholesterol and low-density lipoprotein (LDL) levels are among the most significant and determining variables in the etiology of cardiovascular disease, and they have lately been suggested as potential risk factors in the etiology of several malignancies. The spread of malignant lesions is alarmingly accelerated in several forms of malignancies, such as oral tumours, when cholesterol levels are reduced [23].

The objective of the current study was to assess the serum lipid profile, which included total cholesterol (TC), triacylglycerides (TAG), High-density lipoprotein (HDL), low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) in various cancer patients.

Material and Methods:

Study Design and Ethical Clearance

The Department of Biochemistry at Santosh Medical College, Ghaziabad, Delhi NCR, India, where this study was conceptualized and designed. The institutional ethics committee gave its approval to this investigation.

Data Collection

50 diagnosed subjects (cancer patients) and 50 healthy control subjects' samples reports (Lipid Profile) estimated in fullyautometic analyzer (ERBA EM 200) were collected from central clinical laboratory, SKMCH, Muzaffarpur, Bihar, India. The individuals were told of the study's goals and methods, and their signed informed permission was acquired. Patients with cardiovascular problems, nephrotic syndrome, hyperthyroidism, diabetes mellitus, and blood abnormalities were excluded from this study.

Results

As shown in Table 2, there was a significant difference observed between cancer patients and normal subjects the serum cholesterol levels (144.52 ± 45.12 mg/dL and 171.27 ± 32.45 , p=0.004);

serum triglyceride levels ($128.38 \pm 51.91 \text{ mg/dL}$ and $166.31 \pm 74.16 \text{ mg/dL}$, p=0.01); serum HDL cholesterol levels ($33.84 \pm 6.83 \text{ mg/dL}$ and $37.77 \pm 6.20 \text{ mg/dL}$, p=0.01); serum LDL cholesterol levels ($85.00 \pm 38.67 \text{ mg/dL}$ and $99.03 \pm 24.54 \text{ mg/dL}$, p=0.06) and serum VLDL cholesterol levels ($25.67 \pm 10.34 \text{ mg/dL}$ and $33.84 \pm 16.53 \text{ mg/dL}$, p=0.01) respectively.

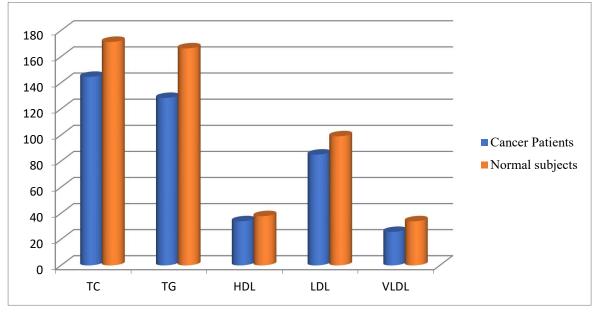
	Cancer Patients (n=50)	Normal subjects (n=50)		
AGE (Year)	51.51 ± 15.23	42.86 ± 14.56		

Table 1. Age distribution [Mean ± SD Age (Years)]

Table 2. Comparison of serum lipid profile level in cancer patients and normal subjects.
[Data represents in Mean ± SD]

	Cancer Patients (n =50)	Normal subjects (n=50)	P- Value
TC (Total	144.52 ± 45.12	171.27 ± 32.45	0.004
Cholesterol) mg/dl	177.32 ± 73.12		
TG (Triglyceride)	128.38 ± 51.91	166.31 ± 74.16	0.01
mg/dl			
HDL (High Density	33.84 ± 6.83	37.77 ± 6.20	0.01
Lipoprotein) mg/dl	55.84 ± 0.85		
LDL (Low Density	85.00 ± 38.67	99.03 ± 24.54	0.06
Lipoprotein) mg/dl	83.00 ± 38.07		
VLDL (Very Low			
Density Lipoprotein)	25.67 ± 10.38	33.84 ± 16.53	0.01
mg/dl			

Graph 1: Comparison of lipid profile in cancer patients and Normal subjects (Mean ± SD)



Discussion:

Lipid profile is linked to both the prognosis and the genesis of cancer. The continuous process of oncogenesis may be the cause of lower blood cholesterol levels in blood compartments and growing tissues. In the current investigation, we assessed the blood levels of lipids in patients with cancer treated with chemotherapy that were related with the most significant prognostic markers.

In the current study we found significant decrease level of serum HDL-cholesterol, LDL-cholesterol, triglyceride, total cholesterol and VLDL-cholesterol. As compared to the normal individuals, other studies have similarly found lower lipid levels in cancer patients, even with varying lipid profile patterns. Lohe et al. [24] suggested that low cholesterol levels in blood compartments and growing tissues may be caused by the carcinogenesis process. The findings of the current study supported those of Ashutosh et al. (2015) [25] and Garg et al. (2014) [26] in that cancer patients had considerably lower blood levels of TC, TG, HDL, LDL, and VLDL when compared to normal values.

According to Patel et al. [27], patients with OSCC had a substantial drop in HDL-cholesterol, VLDL-cholesterol, total cholesterol and TAG but not in LDL-cholesterol. Acharya et al. [28] found that patients with OSCC had a substantial reduction in HDL-cholesterol, LDL-cholesterol, VLDL-cholesterol and total cholesterol but not TAG. The drop in levels of α -lipoprotein and cholesterol noted by Nydegger et al. in 1972 [29] may have been brought on by increased α -lipoprotein and cholesterol catabolism and decreased hepatic production of α -lipoprotein and cholesterol, as the synthesis is influenced by tumor metabolites.

Lipids are essential for many biological functions, including preserving cell integrity, promoting normal and malignant cell growth, and cell division. They are the most important part of a cell membrane. To explain the link between low cholesterol and cancer, there are three primary opposing explanations. (A) Low cholesterol may signal the development of cancer even before it shows up clinically. (b) Low cholesterol acts as a marker for certain other causative sets of factors, and even while it may precede cancer, its link with mouth cancer may be incidental. (c) Low cholesterol levels have been causally linked to several types of cancer and may occur before the onset of cancer [30].

According to a research by Fu-Chuan Chao et al. [31], tumor cells directly use cholesterol for their own metabolism, which causes hypolipidemia as a direct side consequence of this. Recognizing the severe effects of hypolipidemia and taking prompt action to raise cholesterol levels will help prevent circumstances that might increase cancer patients' risk of morbidity and death. It was indicated in a different research by Min-Ah Choi et al. [32] that hypocholesterolemia was a result of low blood levels of antioxidant vitamins. Reduced blood levels of antioxidant vitamins cause the production of more free radicals, which in turn causes an increase in lipid peroxidation.

According to S Desai et al. [33] hypothesis's, free cholesterol in tumor cells is preferentially directed into storage as cholesterol esters rather than being released to the bloodstream as HDL. This occurs regardless of how the free cholesterol enters the tumor cells—through synthesis or absorption. This process explains why cancer patients have lower HDL levels. Low HDL has also

been suggested to be a further predictor of cancer in several studies, and it may be a side effect of a condition where cholesterol is used for membrane biogenesis [34]. Lower HDL levels may also be an indication of early neoplastic and precancerous changes. In the current study, patients in the study group had significantly lower serum HDL levels than those in the control group. Mujoo et al. [35], Anand et al. [36], Meisel et al. [37], and Granero Fernandez et al. [38] all found similar findings in their research to evaluate changes in lipid profile levels.

Conclusion:

In our investigation, we found that, compared to the control group, patients with cancer had considerably lower blood levels of triglycerides, HDL, LDL, and VLDL. This decrease may be attributed to the major alterations in cell integrity. However, a thorough examination of cholesterol-carrying lipoprotein transport and the effectiveness of the receptor system may be useful in figuring out the underlying processes that control the level of plasma cholesterol in cancer. This is a brief study, and further research is needed to the lipid profile be examined in patients with precancerous diseases and chemotherapy treatments in order to draw a firm conclusion.

Conflicts of Interest

None

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