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Original Research Article

Study on the Correlation between Hyperlipidemia and Lipoma

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Abstract:

Introduction: This study explores lipomas, benign tumors arising from fat cells. Lipomas, commonly found on the trunk, are usually harmless and manifest as painless lumps. While the exact cause is uncertain, potential links to stress-induced cytokines and genetic factors are explored. Lipomas, often visible, are removed for aesthetic reasons or if they affect organ function. The study also delves into familial hypercholesterolemia and Multiple Symmetric Lipomatosis, revealing associations between lipoprotein patterns, adipose tissue lipoprotein lipase activity, and abnormal lipid metabolism. The findings provide insights into lipoma development and associated genetic and metabolic factors.

Aims and Objective: To find out whether the lipid profile parameters are associated with the occurrence of lipoma.

Method: This observational study, conducted from September 2022 to August 2023, focused on lipoma patients and a control group of healthy individuals. Baseline characteristics, lifestyle factors, and lipid profiles were analyzed. Inclusion criteria involved patients with completed tests and no underlying chronic conditions, while exclusion criteria included incomplete data. Statistical analysis, performed using SPSS 27, employed ANOVA and t-tests, with a significance level of $P<0.05$. The study aimed to identify associations between lipomas and various parameters, providing valuable insights into potential risk factors.

Results: The study revealed gender distribution differences between the Control and Lipoma groups, indicating a potential association between gender and lipomas. Lifestyle factors, including higher BMI, trauma history, and lower physical activity, were linked to lipomas. Lipoma patients showed elevated cholesterol, particularly LDL, and triglyceride levels compared to controls, suggesting a potential role of lipid metabolism in lipoma development. The findings underscore the need for further exploration into gender-specific risk factors and lipid metabolism's involvement in lipomas.

Conclusion: The study concluded that mainly the elevated triglyceride levels was associated with occurrence of lipoma.

Keywords: Lipoma, Lipid, Lipomatosis, Hyperlipidaemia.

Introduction

Adipocytes, or fat cells, can develop benign tumors called lipomas, which appear as tender, painless lumps that can appear anywhere on the body but are most commonly found on the trunk. Lipomas usually measure one to ten centimeters. Wherever in the body where healthy fat cells have developed is where these mesenchymal tumors can be detected [1]. They have several histologic subtypes and are benign. Many different conditions can manifest as the presence of numerous lipomas. While lipomas are often found in subcutaneous planes that they might occasionally include deeper muscle planes or fascia [2].

The most prevalent mesenchymal tumors in humans are called lipomas. At some point out their lives, 1 in every 1,000 people will develop lipoma. Adipocytes can be found anywhere on the body, although the most frequent locations are the upper extremities and trunk.for lipomas [3]. It is uncertain what specifically causes lipomas. According to one theory, stress causes cytokines to be released, which in turn cause preadipocyte differentiation & maturation. This suggests a possible connection between trauma and the development of lipomas [4]. A small percentage of patients—between 2% and 3%—have numerous lesions that are inherited in a family manner, suggesting that genetics may be involved in certain cases. Some solitary lipomas have been linked to a gene on chromosome 12, and some of these tumors have a variant in the HMGA2-LPP combination gene. Liposomes are also a

clinical symptom of a number of hereditary disorders. Patients with diabetes mellitus, hyperlipidemia, and obesity also have higher incidences of lipomas [5].

Patients frequently report feeling a soft, moveable mass of tissue beneath their skin. Unless they invade blood vessels, joints, or nerves, they are usually harmless. These are often observed by patients in the upper body. These lipomas might sporadically develop in the organs or muscles. The majority of lipomas are benign; they are only surgically removed or treated if they impair an organ's ability to function or if their location causes discomfort [6]. Nonetheless, since these tumors are frequently visible through the skin when they are subcutaneous, for aesthetic reasons, some patients choose to have them removed. Tiny cuts can be utilized to remove lipomas less than 4 cm, and scarring is typically not a major problem. Additionally, studies showed that open surgery, which permits better judgment, avoids damage to surrounding tissues, and prevents recurrences, is still preferable to suction-assisted lipectomy for the removal of giant lipomas (larger than 10 cm). Lesions containing fat called pericallosal lipomas are most frequently found in the interhemispheric fissure, which is strongly linked to the defective corpus callosum. This is where intracranial lipomas are most frequently found [7].

A genetic lipid metabolic illness that is autosomal dominant, familial hypercholesterolemia (FH) usually inherited. It is brought on by a chromosome 19 abnormality and is identified by elevated levels in LDL-C, or low-density lipoprotein cholesterol, is occasionally referred to as "bad cholesterol," which is inherited in families [8]. The illness can result in heart attacks in young children and is present from birth. Clinically, FH is typified by extravascular xanthomas (particularly in the skin as well as tendons of individuals with severe hyperlipidemia) and vascular wall cholesterol deposits that result in atheromas [9].

Fifteen individuals with Multiple Symmetric Lipomatosis (MSL) were examined for serum lipoprotein content and composition, lipoprotein lipase activity in lipomatous tissue, and post-heparin particle lipase functions in plasma. Individuals suffering with MSL had significantly higher levels of activity for Their adipose tissue contains lipoprotein lipase [10]. There was a slight but significant increase in extrahepatic lipolytic activity, but total & liver Plasma lipolytic activity of enzymes following heparin was normal. It was shown that there was an aberrant makeup of serum lipoproteins, with a notable rise in apoprotein A-I and high density lipoproteins, including HDL2 subfraction. It was discovered that low density lipoproteins had an aberrant composition and were declining concurrently [11]. There is a recently identified kind of hyperalphalipoproteinemia that is compatible with this lipoprotein pattern. Profound associations were discovered between adipose tissue lipoprotein lipase activity and blood HDL2 cholesterol levels (as also between the levels of HDL2 cholesterol and serum VLDL-triglycerides) [12]. These results validate fat lipoprotein lipase's role for the degradation of triglyceride-rich lipoproteins. Together with a previously shown reduction in adrenergic-stimulated lipid recruitment, the higher levels that lipoprotein lipase activity with adipose tissue may explain the aberrant fat buildup in lipomatous fat cells as well as the hyperalphalipoproteinemia seen in MSL patients. Due to the fact that MSL affects two brothers, it is believed to be a "triglyceride buildup disease inside adipose tissue" caused by a hereditary enzymatic deficiency [13].

Methods

Study Design: This is an observational study which was conducted on patients who were presented with lipoma in our department. The study considered 2 groups, namely, lipoma group comprising of patients presented with lipoma and another group (Control group) comprising of healthy individuals. This study was conducted from September 2022 to August 2023. In order to conduct this study, it was collected data from baseline characteristic such as age, body mass index (BMI), gender distribution, intensity of physical activity, various histories like trauma history, smoking, alcohol use, surgery, diabetes, and familial lipomatosis. The patients underwent a lipid profile test (including total cholesterol, low-density lipoprotein, high-density lipoprotein, and triglycerides) and the results were analyzed. Each baseline parameter and lipid profile test parameter was statistically analyzed between the patients of the Lipoma group and control group.

Inclusion and Exclusion Criteria

Inclusion Criteria

- The patient who visited our department
- The patient who did all the required tests
- The patient who had no other underlying chronic condition.

Exclusion Criteria

- The patient who could not give the details of his or her history
- The patient whose data was not consistent
- The patient whose any of the lipid profile test parameters was missing.

Statistical Analysis: The study used SPSS 27 for effective statistical analysis. The authors employed ANOVA for analyzing the variables between the groups. The discrete data was expressed as frequency and their respective percentages while the continuous data was written in form of average and

standard deviation. The study used t-test for discrete data analysis while the continuous data was analyzed using ANOVA. The level of significance was considered to be P<0.05.

Results

Figure 1 shows the gender distribution in the Control (n = 25) and Lipoma (n = 25) groups. In the Control group, 14 were male (56%), and 11 were female (44%). There were 18 men (72%) and 7 females (28%), but, in the Lipoma group, the gender ratio was different. Figure 1 shows that there may be a difference between the two groups according to gender; the Control group had more females than males, whereas the Lipoma group had more males. The difference in gender distribution did not reach statistical significance (p = 0.059), but the trend suggests a possible association between gender and lipomas, emphasising the need for further research into gender-specific lipomatosis risk factors.

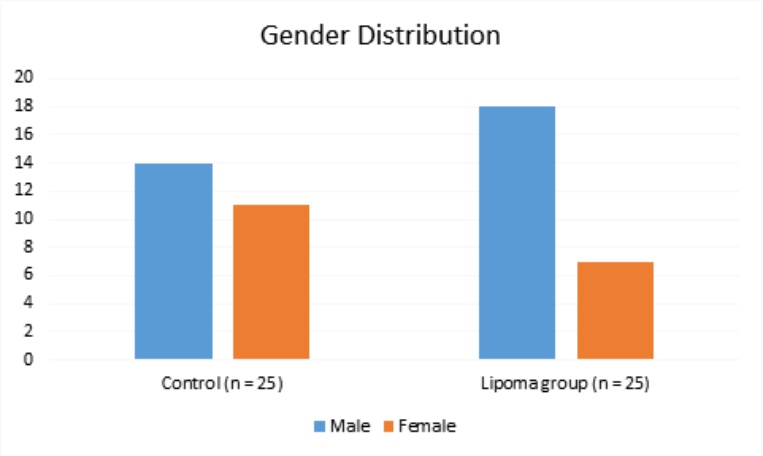

Figure 1: Gender Distribution of the study

Table 1 compares the baseline characteristics of the Control (n = 25) and Lipoma (n = 25) groups. The Control group had a mean age of 48.59±5.96, whereas the Lipoma group had a slightly higher mean age of 52.62±3.65, with a p-value of 0.069, indicating a non-significant age difference. The Lipoma group had a substantially higher BMI (25.47±1.36) than the Control group (22.85±1.12), with a p-value of 0.048. The Lipoma group had 72% females compared to 44% in the Control group, although the p-value of 0.059 shows a borderline significance. The Lipoma group had a higher trauma history (64% vs. 12%, p = 0.0412) and lower moderate to severe physical activity (24% vs. 64%, p = 0.047). Smoking history, alcohol consumption, surgery, diabetes, and familiar lipomatosis also differed between groups, but not statistically. These data show lifestyle factors may be linked to lipomas, warranting additional study.

Table 1: Basline characteristics of the patients in each group

Parameter	Control (n = 25)	Lipoma group (n = 25)	P-value
Age	48.59±5.96	52.62±3.65	0.069
BMI	22.85±1.12	25.47±1.36	0.048
Gender Male Female	 14 (56%) 11 (44%)	 18 (72%) 7 (28%)	0.059
Moderate to Severe Physical Activities	16 (64%)	6 (24%)	0.047
History of trauma	3 (12%)	16 (64%)	0.0412
History of Smoking	12 (48%)	15 (60%)	0.069
Mild to Moderate Alcohol consumption	15 (60%)	16 (64%)	0.069
History of surgery	2 (8%)	19 (76%)	0.0462
History of diabetes	12 (48%)	13 (52%)	0.085
History of familiar Lipomatosis	1 (4%)	14 (56%)	0.0495

Table 2 shows the lipid profiles of 25 Control and 25 Lipoma patients. Mean total cholesterol levels were 155.83±33.8 in the Control group and 180.26±28.45 in the Lipoma group, although the difference was insignificant (p-value = 0.0954). The Lipoma group had significantly higher mean LDL cholesterol levels (145.65±25.44) than the Control group (97.22±4.73), with a p-value of 0.044, showing a link between raised cholesterol and lipomas. Additionally, the Lipoma group had lower HDL

cholesterol levels (37.41±2.98) than the Control group (44.11±5.1), however, the difference was not significant (p = 0.069). Triglyceride (TG) levels were substantially higher in the Lipoma group (311.25±35.15) compared to the Control group (128.45±5.95), with a p-value of 0.012. These data imply that lipomas may be caused by lipid metabolism, as LDL cholesterol and triglycerides may be greater in lipomas. These connections and their processes may require further study.

Table 2: Lipid Profile of the patients in each group

Parameter	Control (n = 25)	Lipoma group (n = 25)	P-value*
Total Cholesterol (mg/dL)	155.83±33.8	180.26±28.45	0.0954
LDL (mg/dL)	97.22±4.73	145.65±25.44	0.044
HDL (mg/dL)	44.11±5.1	37.41±2.98	0.069
TG (mg/dL)	128.45±5.95	311.25±35.15	0.012

*ANOVA at the level of significance, α=0.05

Figure 2 compares Total Cholesterol, LDL, HDL, and Triglycerides between the Lipoma and Control groups. TG (182.8 mg/dL higher in the Lipoma group) and Total Cholesterol (24.43 mg/dL higher in the Lipoma group) are the mean differences. These numerical discrepancies show that the Lipoma group had greater Total Cholesterol, LDL, and TG and lower HDL than the Control group. Positive Total Cholesterol, LDL, and TG differences show a link between increased lipids and lipomas. The Lipoma group has lower HDL levels, which may be worth investigating. Figure 2 emphasises the importance of lipid metabolism in lipomas and quantifies the lipid level differences between the two groups. More research is needed to understand how these lipid profile changes may contribute to lipomas.

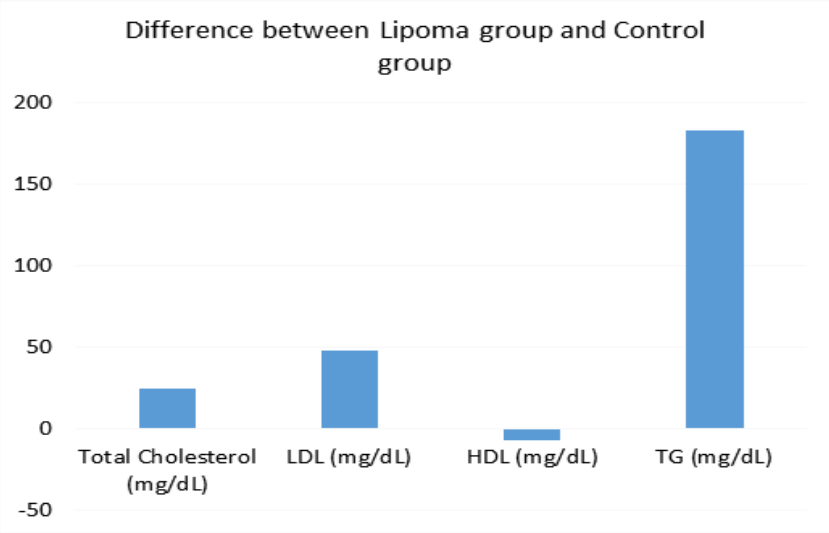

Figure 2: Difference of lipid levels between Lipoma group and Control group

Discussion

A study describes a family characterised with familial combination hyperlipidemia, in which the affected individuals had nonsymmetric subcutaneous lipomatosis (NSSCL). Impacted individuals exhibited low-density lipoprotein (LDL) cholesterol, elevated levels of total cholesterol, and high-density lipoprotein (HDL) cholesterol in their bloodstream. In contrast, family members who did not have non-specific systemic connective tissue lesions (NSSCL) exhibited normal lipid levels [14]. A link was observed between the severity of hyperlipidemia and the quantity of subcutaneous lipomas. The presence of hyperlipidemia in family members using non-small cell lung cancer (NSSCL) indicates a genetic connection between these two traits, however, no correlation was found with HLA haplotyping. This correlation between lipid problems and non-small cell lung cancer (NSSCL) has not been documented before, as far as we know [15].

Homozygous familial hypercholesterolemia is a genetic condition where there is a dominant inheritance pattern and it affects how the body processes lipids. It is characterised by a decreased ability to remove low-density lipoprotein-cholesterol from the bloodstream, which leads to a greater likelihood of developing cardiovascular problems quickly. The occurrence of this condition is relatively infrequent and is estimated to be one in a million in the general population [16].

Another study examined the activity of lipoprotein lipase in lipomatous tissue, the activity of post-heparin lipoprotein lipase in plasma, and the composition and concentration of lipoproteins in the serum of 15 individuals with Multiple Symmetric Lipomatosis (MSL). MSL patients exhibited significantly heightened lipoprotein lipase activity in their adipose tissue. The overall lipolytic activity in the blood and liver after heparin administration was within normal limits. However, there was a noticeable but statistically significant increase in lipolytic activity outside of the liver. A deviation in the composition of lipoproteins in the blood serum was observed, marked by a notable elevation in high density lipoproteins, specifically the HDL2 subfraction, and apoprotein A-I. The heightened levels of lipoprotein lipase activity in adipose tissue, along with a previously established reduction in adrenergic-stimulated lipid mobilisation, may explain the aberrant fat buildup in lipomatous fat cells and the occurrence of hyperalphalipoproteinemia in MSL patients [17].

The lipid makeup of lipomas was examined in relation to various forms of liposarcomas. The primary constituents of lipomas and liposarcomas resembling lipomas were triglycerides [18]. In addition to having a high water content, myxoid liposarcomas also included significant levels on free cholesterol & phospholipids [19,20]. In terms of its lipid composition & water content, one pleomorphic liposarcoma resemble a myxoid liposarcoma. The lipid makeup of four mixed-type liposarcomas was similar to that of the primary subtype. All lipoma-like (highly developed) liposarcomas included a glycerol ether-like percentage in one myxoid type though not in the others [21,22].

The formation of uncapsulated masses for adipose tissue is a characteristic of the uncommon condition known as multiple symmetric lipomatosis (MSL). MSL is linked to heavy ethanol use and is exacerbated by mediastinal adipose tissue infiltration, somatic and autonomic neuropathy, and other factors [23]. Although there are currently insufficient long-term longitudinal data, the condition is thought to proceed slowly. This research presents the long-term inquiries of a sizable number of patients with MSL. Significant morbidity and death are linked to MSL. Consequently, it is impossible to defend the concept of "benign symmetric lipomatosis," which is still used by a number of authors [24].

Another condition Multiple Symmetric Lipomatosis (MSL), an uncommon condition primarily affecting adipose tissue and characterized by the presence of not-encapsulated fat masses symmetrically disposed during characteristic locations on the body (neck, trunk, proximal regions of upper and lower limbs), a revision of our case series (72 patients) and available literature are the goals of the study. It is believed that a malfunction in the development & maturation of human BAT cells causes MSL [25].

To ascertain the spontaneous spinal epidural lipomatosis (SEL) clinical features. Our findings imply that individuals with LSS who have idiopathic SEL possess more intense pain than those without SEL, and that abnormal lipid metabolism may play a role in the pathophysiology of idiopathic SEL [26].

Conclusion

The study concluded that mainly the elevated triglyceride levels was associated with occurrence of lipoma. Also, the study pointed out that the occurrence of lipoma is associated with increased LDL. The lipid profile analysis indicated that the lipoma group had significantly higher mean LDL cholesterol levels and triglyceride levels, suggesting a potential link between elevated cholesterol and lipomas. Total cholesterol levels and HDL cholesterol levels also displayed numerical differences, further emphasizing the involvement of lipid metabolism in lipomas. The visual representation in Figure 2 underscored the lipid level disparities between the lipoma and control groups, with positive differences in total cholesterol, LDL, and triglycerides. These findings suggest a potential association between lipid metabolism alterations and the development of lipomas, calling for further investigation into the underlying mechanisms.

This current study explored the gender distribution, baseline characteristics, and lipid profiles in patients with lipomas compared to a control group. While the gender distribution did not reach statistical significance, a notable trend suggested a possible association between gender and lipomas, emphasizing the need for further research into gender-specific lipomatosis risk factors. The baseline characteristics revealed that the lipoma group had a higher BMI, a higher prevalence of trauma history, and lower engagement in moderate to severe physical activities. Lifestyle factors, including smoking history, alcohol consumption, surgery, diabetes, and familial lipomatosis, also showed variations between groups, warranting additional study.

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