

ORIGINAL RESEARCH

Evaluation of Serum Lipid Levels and the Risk of Venous Thrombosis

¹Dr. Rambir Singh, ²Dr. Dinesh Singh Meena, ³Dr. Neeraj Kumar, ⁴Dr. Sanjiv Singh Chaudhary

^{1,4}Assistant Professor, ³Associate Professor, Department of Surgery, SJP Medical College, Bharatpur, India

²Assistant Professor, Department of Surgery, Medical College Dausa, India

Corresponding Author

Dr. Sanjiv Singh Chaudhary

Assistant Professor, Department of Surgery, SJP Medical College, Bharatpur, India

Received Date: 21 August, 2024

Accepted Date: 25 September, 2024

ABSTRACT

Background: Venous thrombosis can manifest as either superficial venous thrombosis or deep venous thrombosis (DVT). Deranged lipid profile is also one of the risk factors responsible for Venous Thrombosis. Hence; the present study was conducted for assessing Serum Lipid Levels and the Risk of Venous Thrombosis. **Materials & methods:** A total of 50 healthy controls and 50 subjects with presence of venous thrombosis were enrolled. Complete demographic and clinical details of all the patients was obtained. Blood samples were obtained in the morning and serum lipid profile was evaluated. Serum levels of cholesterol and triglycerides were assessed using a standardized enzymatic procedure. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software. **Results:** Mean age of the patients of the control group and study group was 50.9 years and 53.1 years respectively. Majority proportion of subjects of both the study groups were males. Among study group, diabetes, positive smoking habit and hypertension was seen in 30 percent, 32 percent and 26 percent of the patients respectively. Significantly lower levels of LDL-cholesterol levels were seen among subjects of the study group. Dyslipidemia was seen in higher frequency among subjects of the study group in comparison to the control group. **Conclusion:** Deranged lipid profile is a significant risk factor for occurrence of venous thrombosis. Hence; timely screening of lipid profile should be done.

Key words: Lipid, Deranged, Venous thrombosis

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-Non Commercial-Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

INTRODUCTION

The pathophysiology of venous thrombosis has been extensively articulated by Rudolf Virchow, encapsulated in what is known as Virchow's triad, which comprises stasis, endothelial injury, and hypercoagulability. Venous thrombosis can manifest as either superficial venous thrombosis or deep venous thrombosis (DVT). Although the majority of DVT cases originate in the extremities, particularly in the lower limbs compared to the upper limbs, thrombotic events can also arise in other vascular regions, including the mesentery, pelvis, cerebral circulation, and portal tract. DVTs can lead to significant morbidity, particularly due to post-thrombotic syndrome, which results in local tissue damage. The most serious complication, which carries a high mortality rate, is pulmonary embolism (PE) that arises from venous thromboembolism (VTE).¹⁻³ Numerous risk factors, both genetic and environmental, have been thoroughly investigated and linked to venous thrombotic events. Recognizing these risk factors can enhance diagnostic methods and, crucially, aid in the prevention of thrombotic occurrences. Preventive measures, such as the

application of pneumatic devices and the use of prophylactic anticoagulation, are considered standard practice in hospital settings. These strategies are informed by the identification of individual patient risk factors.^{4, 5} The presence of antiphospholipid antibodies (APLA) is associated with an increased risk of arterial and venous thrombosis involving any organ system. Deranged lipid profile is also one of the risk factors responsible for Venous Thrombosis.^{6, 7} Hence; the present study was conducted for assessing Serum Lipid Levels and the Risk of Venous Thrombosis.

MATERIALS & METHODS

The present study was conducted for Serum Lipid Levels and the Risk of Venous Thrombosis. A total of 50 healthy controls and 50 subjects with presence of venous thrombosis were enrolled. Complete demographic and clinical details of all the patients was obtained. Blood samples were obtained in the morning and serum lipid profile was evaluated. Serum levels of cholesterol and triglycerides were assessed using a standardized enzymatic procedure. The measurement of cholesterol within the HDL fraction

was conducted through a direct enzymatic method aimed at quantitatively determining HDL cholesterol in human serum. LDL cholesterol values were calculated according to the Friedewald formula, while non-HDL cholesterol was derived by subtracting HDL cholesterol from total cholesterol. Additionally, the atherosclerosis index, defined as the ratio of LDL cholesterol to HDL cholesterol, and various atherogenic ratios, including total cholesterol to HDL cholesterol and non-HDL cholesterol to HDL cholesterol, were computed. Patients with presence of any other systemic illness or any known drug allergy were excluded. All the results were recorded in Microsoft excel sheet and were subjected to statistical analysis using SPSS software.

RESULTS

Mean age of the patients of the control group and study group was 50.9 years and 53.1 years respectively. Majority proportion of subjects of both the study groups were males. Among study group, diabetes, positive smoking habit and hypertension was seen in 30 percent, 32 percent and 26 percent of the patients respectively. Significantly lower levels of LDL-cholesterol levels were seen among subjects of the study group. Dyslipidemia was seen in higher frequency among subjects of the study group in comparison to the control group.

Table 1: Clinical variables

Variable	Control group	Study group
Mean age (years)	50.9	53.1
Males	29	30
Females	21	20
Diabetes	12	15
Smoking	10	16
Hypertension	8	13

Table 2: Lipid profile

Lipid profile	Control group	Study group	p-value
Total cholesterol (mmol/L)	5.66	5.81	0.19
Triglycerides (mmol/L)	1.55	1.51	0.58
LDL-cholesterol (mmol/L)	3.91	3.25	0.00*
HDL-cholesterol (mmol/L)	1.23	1.29	0.18

*: Significant

Table 3: Incidence of dyslipidemia

Dyslipidemia	Control group	Study group	p-value
Number	2	7	0.00*
Percentage	4	14	

*: Significant

DISCUSSION

Venous thrombosis and arterial cardiovascular disease have historically been viewed as distinct entities, each with unique etiologies and therapeutic approaches. Recent research over the last ten years, however, has indicated that individuals suffering from venous thrombosis, such as deep vein thrombosis or pulmonary embolism, exhibit a heightened risk for subsequent arterial conditions. Given that lipid levels can be influenced through lifestyle modifications and pharmacological interventions, the potential link between lipids and venous thrombosis, along with its underlying pathophysiological mechanisms, represents a significant clinical concern that warrants further investigation. Notably, existing evidence suggests that lipid-lowering medications, particularly statins like rosuvastatin, may correlate with a reduced incidence of venous thrombosis, hinting at a possible involvement of lipids in the pathophysiology of this condition. Nevertheless, the precise relationship

between lipids and venous thrombosis remains inadequately understood, largely due to conflicting findings in epidemiological research.⁹⁻¹¹ Venous thrombosis (VT) represents a prevalent medical condition, characterized by a significant five-year cumulative recurrence rate that ranges between 12% and 30%, depending on the study. This situation presents a challenge for both clinicians and patients, as the cessation of anticoagulant therapy could lead to the occurrence of a new thrombotic episode, while the continuation of oral anticoagulation carries a risk of major bleeding, estimated at 1–3% annually. Therefore, understanding the risk factors associated with recurrent VT is essential, as it can inform clinical decisions regarding the appropriate duration of anticoagulation following an initial VT event.¹⁰⁻¹²

Mean age of the patients of the control group and study group was 50.9 years and 53.1 years respectively. Majority proportion of subjects of both the study groups were males. Among study group,

diabetes, positive smoking habit and hypertension was seen in 30 percent, 32 percent and 26 percent of the patients respectively. Significantly lower levels of LDL-cholesterol levels were seen among subjects of the study group. Dyslipidemia was seen in higher frequency among subjects of the study group in comparison to the control group. Spasić I et al investigated the influence of lipid metabolism disorders on the risk of deep vein thrombosis. A total of 200 subjects participated in the study, 100 of whom experienced DVT with or without PTE, and 100 healthy subjects representing the control group. The analysis was adjusted for all potential confounders (age, sex, obesity) related to the functionality of the lipid metabolism, and at the same time, may have an impact on the risk of venous thrombosis. The results of the comparison of the mean values of individual lipid status parameters between the patient group and the control group clearly indicate higher concentrations of total cholesterol, total triglycerides, and LDL-cholesterol in the patient group relative to the control group, with a statistically significant difference observed only in the case of LDL-cholesterol concentrations. They have found that type IIa hyperlipoproteinemia is associated with a nearly double increased risk for deep vein thrombosis, while type IIb, IV, or hyperLp (a) lipoproteinemia did not influence the risk. Hypercholesterolemia doubles the risk of deep vein thrombosis development.¹²

CONCLUSION

Deranged lipid profile is a significant risk factor for occurrence of venous thrombosis. Hence; timely screening of lipid profile should be done.

REFERENCES

1. Rosendaal FR. Risk factors for venous thrombosis: prevalence, risk, and interaction. *Semin Hematol.* 1997; 34: 171–187.
2. Sofi F, Marcucci R, Abbate R, Gensini G F, Prisco D. Lipoprotein (a) and Venous Thromboembolism in Adults: A Meta-Analysis. *Am J Med.* 2007;120(8):728.
3. Mccoll M D, Sattar N, Ellison J, Tait R C, Walker I D, Packard C J, et al. Lipoprotein(a), cholesterol and triglycerides in women with venous thromboembolism. *Blood Coagul Fibrinolysis.* 2000;11:225–234.
4. Tsai A W, Cushman M, Rosamond W D, Heckbert S R, Polak J F, Folsom A R. Cardiovascular risk factors and venous thromboembolism incidence: The longitudinal investigation of thromboembolism etiology. *Arch Intern Med.* 2002;162(10):1182.
5. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. *Nature.* 1993; 362: 801–809.
6. Rosenson RS, Lowe GD. Effects of lipids and lipoproteins on thrombosis and rheology. *Atherosclerosis.* 1998; 140: 271–280.
7. Griffin JH, Fernandez JA, Deguchi H. Plasma lipoproteins, hemostasis and thrombosis. *ThrombHaem.* 2001; 86: 386–394.
8. Heit J A, Mohr D N, Silverstein M D, Petterson T M, O'fallon M W, Melton J L. Predictors of recurrence after deep vein thrombosis and pulmonary embolism: A population based cohort study. *Arch Intern Med.* 2000;160(6):761.
9. Lijfering WM, Flinterman LE, Vandenbroucke JP, et al. Relationship between venous and arterial thrombosis: a review of the literature from a causal perspective. *Semin ThrombHemost.* 2011;37(8):885–896
10. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Third Report of the National Cholesterol Education Program (NCEP) Expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III) final report. *Circulation.* 2002;106(25):3143–3421.
11. Glynn RJ, Danielson E, Fonseca FA, et al. A randomized trial of rosuvastatin in the prevention of venous thromboembolism. *N Engl J Med.* 2009;360(18):1851–1861.
12. Spasić I et al. Influence of lipid metabolism disorders on venous thrombosis risk. *J Med Biochem.* 2021 Jun 5; 40(3): 245-251.