



A STUDY OF FASTING LIPID PROFILE IN PATIENTS WITH STROKE AT DR. VITTHALRAO VIKHE PATIL PRAVARA RURAL HOSPITAL, LONI.

¹Dr. Rushikesh C Banale (Post-graduate, General Medicine)

²Dr. Sudhir Tungikar (Professor, General Medicine)

¹PG Student, DR. Vitthalrao Vikhe Patil Pravara Rural Hospital, Loni.

²Professor, DR. Vitthalrao Vikhe Patil Pravara Rural Hospital, Loni.

Abstract: Introduction: Stroke is a global health problem. It is the leading cause of disability and the second leading cause of mortality worldwide. Dyslipidemia is been considered to be a risk factor for stroke, Present study is intended to know association of serum lipid profile with stroke. **Objectives:** To determine the prevalence of abnormal fasting lipid profile including extended lipid profile in patients with stroke. **Results:** A total of 200 patients with stroke were studied. Among the studied patients 112 (56%) were males and 88 (44%) were females. Mean age of the study population was 56.98yrs (SD = 13.8). Out of 200, 108 (54%) were having total cholesterol value <200mg/dl and 92 (46%) were having > 200 mg/dl. 86(43%) subjects were having Triglyceride level above 150 mg/dl, whereas 114 (57%) were having less than 150 mg/dl. 129 (64.5%) were having HDL value less than 40mg/dl whereas 71 (35.5%) were having HDL > 40 mg/dl. 106 (53%) were having VLDL value < 30mg/dl whereas 94 (47%) were having VLDL>30 mg/dl. 159 (79.5%) were having LDL values above 100 mg/dl and 41 (20.5%) were having LDL value less than 100mg/dl. 74 (37%) were having Apolipoprotein-A1 value less than 120 mg/dl and 126 (63%) were having value > 120 mg/dl. 126 (63%) were having Apolipoprotein-B value <130 mg/dl and 74 (37%) were having value > 130 mg/dl. There was no statistically significant difference among males and females in the study subjects. There was statistically significant positive correlation between Total cholesterol & LDL with Apolipoprotein-B (p < 0.001). **Conclusion:** In the present study dyslipidemia, low Apolipoprotein-A1 and high Apolipoprotein B have been found to be associated with stroke.

Keywords: Total cholesterol, Triglycerides, HDL, LDL, Apolipoprotein A1, Dyslipidemia, Stroke

Introduction:

Stroke is a global health problem. It is the leading cause of disability and the second leading cause of mortality worldwide.¹ WHO defined stroke as “rapidly developed clinical signs of focal disturbances of cerebral function; lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin”. The 24 hour threshold excludes Transient Ischemic Attacks (TIA).^{2,3}

The incidence and mortality of stroke vary greatly among the different population of world and has declined considerably in several foreign countries probably due to better preventive measurements. According to World Health Organization, stroke is second largest cause of mortality throughout the world. Recent studies conducted on Indian population has shown that age-adjusted prevalence rate of stroke is between 250-350/100,000.^{3,4} In developed countries, more than 80% of cases

of CVA occur in individuals who are above 60yrs of age, whereas in India about 1/5th of the all strokes occur in individuals who are below the age of 60 year.⁵

There are various non modifiable risk factors like age, sex, familial trends, ethnic groups, race and modifiable risk factors like cardiac diseases, diabetes mellitus, dyslipidemia, hypertension, smoking, alcohol abuse & physical inactivity.⁶ Though substantial differences are there in occurrence of frequency from place to place, cerebral thrombosis is the most frequent form of stroke in studies done by clinical researchers followed by hemorrhage. Cerebral hemorrhage and cerebral embolism comes next as regards the mortality and morbidity.³

Abnormal fasting lipid profile results of Total cholesterol (TC), Triglycerides (TG), Low density lipoprotein (LDL) & High Density Lipoprotein (HDL) Cholesterol are considered as risk factors for stroke due to their strong link leading to atherosclerosis. The apolipoprotein are the protein component of the lipoproteins. Apolipoprotein-B (Apo B) which reflects the concentration of potentially atherogenic LDL Apolipoprotein-A1 (ApoA1) which reflects the concentration of antiatherogenic HDL are considered as additional lipoprotein related parameters that indicate a vascular risk.⁷

Dyslipidemia, low Apolipoprotein-A1 and high Apolipoprotein-B are widely accepted as the risk factors for occurrence of Stroke.⁸ Hence the present study was carried out to compare serum lipid profile with extended parameters like Apo A1 & Apo B in patients with stroke.

Objectives Of The Study :

To determine the prevalence of abnormal lipid profile including extended lipid profile in patients with stroke.

Materials And Methods:

Present study is a cross sectional study conducted for a duration of 2 years at DR. VITTHALRAO VIKHE PATIL PRAVARA RURAL HOSPITAL, Loni, Maharashtra. A total of 200 cases were studied.

Fasting blood samples were collected from patients admitted in hospital, and as per the inclusion and exclusion criteria after taking informed consent diagnosed of having stroke based on clinical & CT findings and then their Apolipoprotein-A1 & Apolipoprotein B levels were estimated

Inclusion Criteria

1. The patients aged between 18 and 80 years diagnosed with stroke
2. Both males and females were included in the study.

Exclusion Criteria

1. Congenital heart disease (CHD)
2. Ischemic heart disease (IHD)
3. Atrial fibrillation(AF)
4. Valvular heart disease(VHD)
5. Patients on anticoagulant, antiplatelet drugs and hypolipidemic drugs.

Results:

A total of 200 patients with stroke were studied. Among the 200 patients, 112 (56%) were males and 88 (44%) were females. Mean age of the study population was 56.98yrs (SD = 13.8). 2 (1%) of the patients were aged <20yrs, 5 (2.5%) were aged between 20-30yrs, 12 (6%) were aged 30-40yrs, 30 (15%) were aged between 40 -50yrs, 38 (19%) were aged between 50 – 60yrs, 70 (35%) were aged between 60-70yrs, 34 (17%) were aged between 70-80yrs and 9 (4.5%) were aged more than 80yrs. Mean total cholesterol of the study population was 195.7 mg/dl (SD = 35.43) with minimum value of 84 mg/dl and maximum value of 315mg/dl. Mean triglyceride level of the study patients was 168.4 mg/dl (SD = 91.3) with minimum value of 46mg/dl and maximum value 500 mg/dl. Mean HDL level of the study patients was 37.19 mg/dl (SD =9.04), minimum value of 13 mg/dl and maximum value of 71 mg/dl. Mean VLDL level of the study population was 34.67 mg/dl (SD = 19.7), minimum value was 9 mg/dl and maximum was 114 mg/dl. Mean LDL level of the study population was 125.07 mg/dl (SD = 31.25), minimum value was 11 mg/dl and maximum value was 212 mg/dl. Mean Apolipoprotein-A1 value was 140.3 mg/dl (SD =

39.25), minimum value was 80 mg/dl and maximum value was 202 mg/dl. Mean Apolipoprotein–B was 126.2 mg/dl, minimum value was 72 mg/dl and maximum was 198 mg/dl. (Table 1). Out of the 200 patients studied 108 (54%) were having total cholesterol value 200 mg/dl. 86(43%) subjects were having Triglyceride level above 150 mg/dl, whereas 114 (57%) were having < 150 mg/dl. 129 (64.5%) were having HDL value less than 40mg/dl whereas 71 (35.5%) were having HDL > 40 mg/dl. 106 (53%) were having VLDL value < 30mg/dl whereas 94 (47%) were having VLDL >30 mg/dl. 159 (79.5%) were having LDL values above 100 mg/dl and 41 (20.5%) were having LDL value less than 100mg/dl. 74 (37%) were having Apolipoprotein–A1 value less than 120 mg/dl and 126 (63%) were having value > 120 mg/dl. 126 (63%) were having Apolipoprotein–B value 130 mg/dl. (Table 2)

Table 1: Distribution of study subjects based on their lipid profile values

| Lipid profile | Minimum | Maximum | Mean | Std. Deviation |
|--------------------|---------|---------|--------|----------------|
| TC | 84 | 315 | 195.70 | 35.436 |
| TG | 46 | 500 | 168.40 | 91.364 |
| HDL | 13 | 71 | 37.19 | 9.042 |
| VLDL | 9 | 114 | 34.67 | 19.725 |
| LDL | 11 | 212 | 125.07 | 31.256 |
| Apolipoprotein –A1 | 80 | 202 | 140.30 | 39.252 |
| Apolipoprotein – B | 72 | 198 | 126.20 | 32.548 |

Table 2: Distribution of study subjects based on their lipid profile range

| Lipid profile | Range | Frequency | Percent |
|-------------------|-------------|-----------|---------|
| Total cholesterol | <200 mg/dl | 108 | 54.0 |
| | >200 mg/dl | 92 | 46.0 |
| Triglyceride | < 150 mg/dl | 114 | 57.0 |
| | > 150 mg/dl | 86 | 43.0 |
| HDL | < 40 mg/dl | 129 | 64.5 |
| | >40 mg/dl | 71 | 35.5 |
| VLDL | < 30 mg/dl | 106 | 53.0 |
| | >30 mg/dl | 94 | 47.0 |
| LDL | < 100 mg/dl | 41 | 20.5 |
| | >100 mg/dl | 159 | 79.5 |
| Apolipo –A1 | <120 mg/dl | 74 | 37.0 |
| | > 120mg/dl | 126 | 63.0 |
| Apolipo –B | <130 mg/dl | 126 | 63.0 |
| | > 130mg/dl | 74 | 37.0 |

There was no statistically significant difference in Total cholesterol, Triglyceride, HDL, VLDL, LDL, Apolipoprotein–A1, Apolipoprotein–B among males and females in the study subjects. (Table 3)

| Lipid profile | Sex | N | Mean | Std. Deviation | Std. Error Mean | p value |
|---------------|--------|-----|--------|----------------|-----------------|---------|
| TC | Male | 112 | 196.96 | 37.234 | 3.518 | .575 |
| | Female | 88 | 194.11 | 33.148 | 3.534 | |
| TG | Male | 112 | 170.97 | 89.708 | 8.477 | .654 |
| | Female | 88 | 165.12 | 93.842 | 10.004 | |
| HDL | Male | 112 | 37.12 | 8.771 | .829 | .904 |
| | Female | 88 | 37.27 | 9.425 | 1.005 | |
| VLDL | Male | 112 | 35.55 | 20.160 | 1.905 | .477 |
| | Female | 88 | 33.55 | 19.213 | 2.048 | |
| LDL | Male | 112 | 126.22 | 32.849 | 3.104 | .560 |
| | Female | 88 | 123.62 | 29.224 | 3.115 | |
| APOLIPO-A1 | Male | 112 | 139.78 | 38.886 | 3.674 | .832 |
| | Female | 88 | 140.97 | 39.927 | 4.256 | |
| APOLIPO-B | Male | 112 | 124.84 | 34.312 | 3.242 | .506 |
| | Female | 88 | 127.93 | 30.260 | 3.226 | |

As shown in Table 4, there was statistically significant negative correlation between Total cholesterol & LDL with Apolipoprotein–A1 ($p < 0.001$) (Fig. 1 & 2), There was no statistically significant correlation between TG, HDL, VLDL with Apolipoprotein–A1. There was statistically significant positive correlation between Total cholesterol & LDL with

Apolipoprotein-B ($p < 0.001$) (Fig. 3 & 4), there was no statistically significant correlation between TG, HDL, VLDL with Apolipoprotein-B.

Table 4: Correlation between other lipid profile with Apolipoprotein-A1 & Apolipoprotein-B

| | | TC | TG | HDL | VLDL | LDL |
|--------------------|---------------------|---------|--------|--------|--------|---------|
| Apolipoprotein -A1 | Pearson Correlation | -0.405 | 0.018 | -0.031 | 0.007 | -0.500 |
| | p value | < 0.001 | 0.803 | 0.664 | 0.920 | < 0.001 |
| Apolipoprotein -B | Pearson Correlation | 0.386 | -0.023 | 0.000 | -0.020 | 0.525 |
| | p value | < 0.001 | 0.745 | 0.997 | 0.782 | < 0.001 |

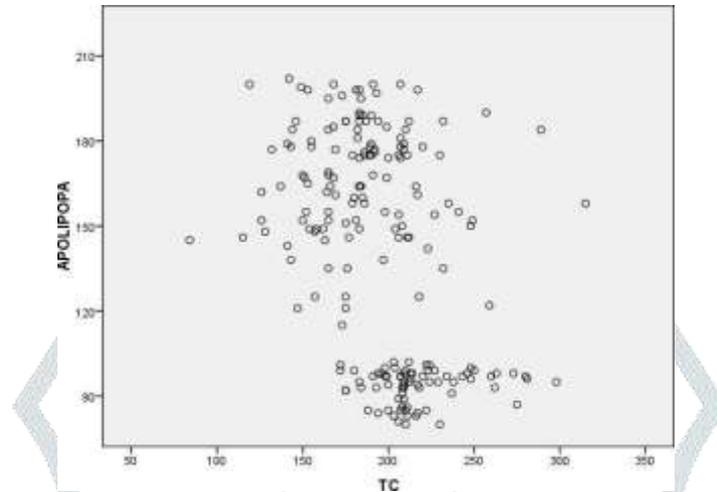


Fig. 1: Scatter diagram showing correlation between total cholesterol & Apolipoprotein - A1

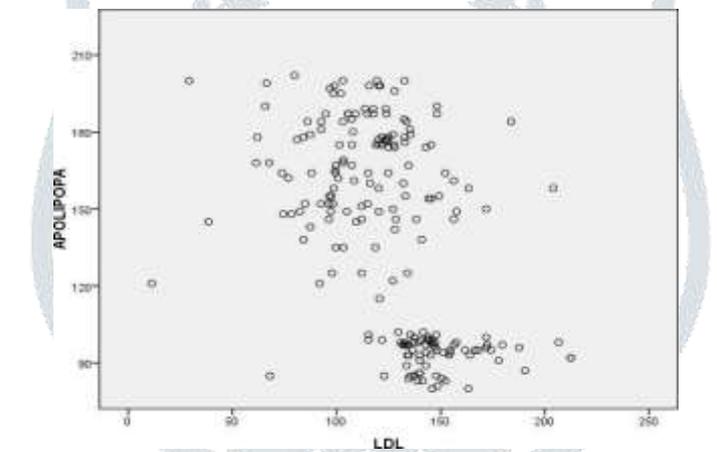


Fig. 2: Scatter diagram showing correlation between LDL & Apolipoprotein-A1

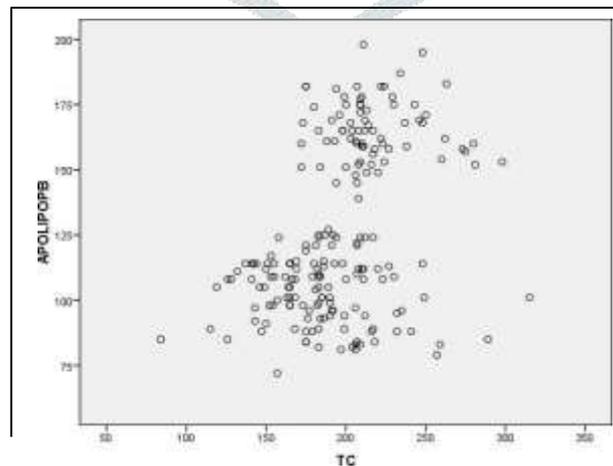


Fig. 3: Scatter diagram showing correlation between Total cholesterol & Apolipoprotein-B

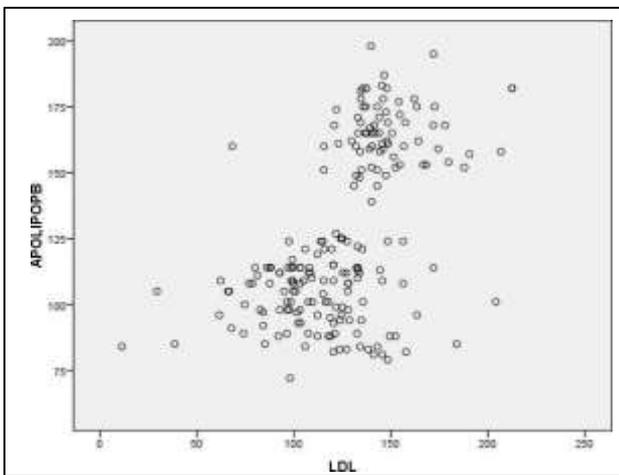


Fig. 4: Scatter diagram showing correlation between LDL & Apolipoprotein-B

Discussion

Dyslipidemia has been described as one of the risk factor for stroke. The incidence and mortality of stroke vary greatly among the different population of world. In the present study a total of 200 cases were studied to know the association of lipid levels in patients with stroke. Out of 200 cases, 112(56%) were males and 88(44%) were females with male: female ratio (M: F) of 1.27:1.

The mean age of presentation was 56.98 ± 13.8 years. In the study done by Mahmood et al⁹ mean age of patients with stroke was 64.2 ± 12 years which was similar to findings of present study he observed male to female ratio of 3.6:1. In another study done by Appelros P et al¹⁰ they found age adjusted rate ratio of M: F was 1.24:1 which was similar to findings in the present study. Amarenco and Steg¹¹ conducted 61 prospective observational studies, and concluded that no association exists between total cholesterol and stroke mortality. They concluded that stroke was a multifactorial disease and that its various causes are not equally associated with blood cholesterol levels.

Study done by Millions et al¹² showed a strong relation between serum total cholesterol and nonhaemorrhagic strokes with an inverse association to intracranial haemorrhage.

Similar findings were observed in the present study.

Study done by Park et al¹³ showed negative results whereas others showed a positive association with high serum triglyceride concentrations.. There is an inverse association between HDL cholesterol and ischemic stroke in the present study as 65.4% patients of ischemic stroke had lower than normal.

Cynthia et al¹⁶ reported that 56% of stroke patients had dyslipidemia, in the present study 79.5% of cases showed similar findings. Cynthia et al also stated that most of them had high triglycerides and low HDL levels, which is in accordance to present study data. Denti et al¹⁷ reported that LDL-C concentrations over 100 mg/dl along with low HDL-C levels were associated with higher risk for stroke, similar findings were observed in present study. Korean Morang et al¹⁸ in their study observed that the mean value of Apo B was higher, that of Apo A1 was lower, similar findings were observed in the present study.

Conclusion

Stroke is a multifactorial disease, there are multiple risk factors involved for the disease occurrence. Dyslipidemia in the form of high Total and LDL cholesterol, low Apo A1 and high Apo B are an important risk factor in the development of stroke. Hence lipid profile assessment is proven to be of help to alert patients. Early recognition and treatment is helpful for reduction in morbidity and mortality.

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