Comparison of Lipid Profile in Thrombotic and Haemorrhagic Stroke Patients

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Objective: To compare lipid profile in thrombotic and haemorrhagic stroke patients. **Place of Study:** All general medical and neurology wards of Mayo Hospital Lahore. **Patients and Methods:** Study was conducted in 40 consecutive patients of stroke admitted in hospital. Serum lipid (total cholesterol, triglycerides, low density lipoproteins, very low density lipoproteins, high density lipoproteins and LDL/HDL ratio) were estimated in all selected patients within 7 days of onset of stroke and findings were entered on study proforma specifically designed for this purpose and analyzed statistically by student's "t" test. **Results:** Out of 40 patients included in the study, 22(55%) were suffering from thrombotic stroke while 18(45%) suffered from haemorragic stroke. The difference in the values of total cholesterol, triglycerides, low-density lipoproteins, very low density lipoproteins, high density lipoproteins and LDL/HDL ratio was not statistically significant between the two groups of patients. **Conclusion:** It is concluded that serum lipids i.e., total cholesterol, triglycerides, low density lipoproteins, very low density lipoproteins, high density lipoproteins and LDL/HDL ratio does not have any differential effect in the causation of thrombotic or haemorrhagic strokes.
Key words: Lipids, thrombotic stroke, haemorrhagic stroke.

Stroke is the third leading cause of death and also a major cause of chronic disability all over the world[1]. Although the treatment of acute stroke has improved, the greatest reduction in stroke mortality and morbidity may possibly be achieved through more effective prevention strategies. To achieve this goal, risk factor identification and where possible modification, is of primary importance. Non-modifiable risk markers for stroke are age, gender, family history, race and ethnicity while modifiable risk factors include hypertension, diabetes mellitus, cardiac diseases (particularly atrial fibrillation), hyperlipidemia, cigarette smoking, alcohol abuse, physical inactivity, asymptomatic carotid stenosis and transient ischaemic attacks[2].

Studies show that three most important modifiable risk factors responsible for stroke are elevated blood pressure, raised serum cholesterol and smoking. These factors are important not only because they increase the risk of stroke but also because they determine the site of vascular pathology.

Hypertension primarily affects small intracranial vessels whereas lipoprotein abnormalities appear to affect mainly the larger cerebral arteries. The role of high serum cholesterol levels in coronary atherosclerosis is established beyond doubt. However, this relationship is less certain as far as stroke is concerned. The conclusions from MRFIT trial[3] have established an inverse relationship between serum cholesterol and risk of death from haemorrhagic stroke. In contrast to this, the deaths from thrombotic stroke and cardiovascular disease were directly related to serum cholesterol[4].

Several acute events e.g. myocardial infarction, severe pain, surgery and even stroke itself are known to affect serum cholesterol level in immediate post-ictus period[5,6]. However, it is still unclear how various types of acute stroke affect the different lipid and lipoprotein levels. They are expected to be different in stroke subtypes because of their varied behavior as risk factor as far as etiopathogenesis of different types of stroke are concerned[4].

These risk factors are amenable to modification by drugs, diet and other interventions[7,8]. Studies show that measurement of levels of lipid at the time of occurrence of stroke may be better representative of usual lipid levels, because poor nutrition or newly developed liver and renal dysfunction after stroke may result in lower cholesterol levels three months later. However, lipid levels measurement at the time of admission have the additional advantage of including both fatal as well as non-fatal strokes[9].

This review was intended to highlight the role of plasma lipids in the pathogenesis of different types of stroke, the importance of their measurement and role of their modification in the prevention of stroke.

Patients and methods:

This study was conducted in all general medical and neurology wards of Mayo Hospital, Lahore which included 40 consecutive patients of stroke who were admitted through out-patients and emergency departments of Mayo Hospital, Lahore.

All male and female patients were diagnosed by using World Health Organization (WHO) criteria for acute stroke i.e., "rapid onset of clinical signs of focal or global disturbances of cerebral function lasting 24 hours or longer or leading to death with no apparent cause other than of vascular origin[10]. Men and women of any age presented with completed stroke within 7 days of onset were *included* while patients with recurrent stroke, transient ischaemic attacks, presented after 7 days of onset were *excluded*. Patients who were having any systemic or cardiac disease likely to generate embolus, alcoholic patients, and patients received any pharmacological agent

liable to affect serum lipids were also excluded. Detailed history of hemiplegia/hemiparesis with or without loss of consciousness was taken from the patient or from the relative and detailed neurological examination was performed to make a clinical diagnosis of thrombotic or haemorrhagic stroke. Computerized Axial Tommographic (CAT) scan brain was performed in all patients under study to confirm the type of stroke.

After confirming the type of stroke on CAT scan, four to five ml of non-citrated venous blood was collected in 5cc plastic disposable syringe between 8-9 a.m after over night fast[11]. After collection, all samples were transported to the department of Pathology, King Edward Medical College, Lahore for serum lipid estimations where each sample was analyzed for:

1. Total cholesterol and triglyceride by enzymatic colorimetric method using standard reagents[12].
2. HDL by precipitation method described by Burstein et al[13].
3. While VLDL and LDL were estimated by using Friedwald Formulae[14].

   VLDL=triglycerides /5

   LDL=total cholesterol – (HDL + VLDL)

All data was entered on study proforma, which was specifically made for this purpose.

Blood glucose, serum electrolytes, complete urinalysis, ECG and X-ray chest were carried out to rule out Diabetes Mellitus, Hypertension, Cardiac abnormalities like atrial fibrillation. Informed consent was taken from each patient under study.All data was analyzed with statistical package for social sciences (SPSS) 10 and independent student's 't' test was applied as test of significance.

Results:

A total of 40 patients with completed stroke were included in this study. Twenty two (55%) had thrombotic stroke and among them, 13 (32.5%) were males and 9 (22.5%) were females while 18 suffered from haemorrhagic stroke and among them, 8 (20%) were males and 10 (25%) were females.

Serum cholesterol was measured in all patients under study. Those with thrombotic stroke had a mean ± SD serum cholesterol of 226.27 ±60.60mg% while patients with haemorrhagic stroke had a mean ±SD value of 202.28 ±63.56 mg%. The difference between the two values was not significant statistically (P value >0.05).

Serum triglyceride levels were 174.61±62.41mg% in patients with haemorrhagic stroke as compared to 164.86±65.33mg% in patients with thrombotic stroke. The difference was not significant statistically (P value <0.05). Similarly the differences between the two groups for serum LDL, HDL, VLDL levels and LDL/HDL ratio were also insignificant from statistical point of view.

Discussion:

The percentage of patients with thrombotic stroke in our study was 55 while that of patients with thrombotic stroke of Okumura K et al[15] was 59.64. The percentage of patients with haemorrhagic stroke in our study was 45 while 40.36% of patients in the study of Okumura K et al[15] suffered from haemorrhagic stroke. This slight difference in proportion of haemorrhagic and thrombotic stroke patients in the two studies can be explained on the basis of racial difference.

The total cholesterol in our patients with haemorrhagic stroke was 202.28 ±63.56 mg/dl. The same value in the study of Garg RK et al[4] was 169.35 ±39.94 mg/dl. The difference in our values and values of Garg RK et al[4] was insignificant statistically whereas the difference between the total cholesterol in patients with thrombotic stroke between the two studies was statistically significant. The levels in our patients being higher than those in patients of Garg RK et al[4](P value >0.05).

Serum triglyceride levels in our patients with haemorrhagic stroke remained 174.61 ±62.41 while in Garg RK et al[4] study it was 146.23 ±102.53 in the similar group. While in thrombotic stroke patients it was 164.86 ±65.33(mg/dl) in our study in comparison to 162.26 ±91.72 in the same group of Garg RK et al[4] study. The difference between values of two studies was statistically insignificant in both types of stroke (P value >0.05).

The low density lipoprotein (LDL) levels in haemorrhagic stroke patients in our study was 118.17 ±45.23 which was 93.29 ±41.27 in the study of Garg RK et al[4] in the similar group of patents. While in thrombotic stroke patients it was 123.91 ±56.18 in our study and 106.02 ±39.90 in the study of Garg RK et al[4]. The difference in our values and values of study of Garg RK et al[4] remained insignificant statistically in both types of stroke (P value >0.05).

High density lipoprotein (HDL) level in haemorrhagic stroke patients was 48.06 ±16.98 in our study while in Garg RK et al[15] study, it was 43.35 ±13.54. In thrombotic stroke patients it was 52.68 ±37.87 in our study as compared to 35.57 ±15.38 in the study of Garg RK et al[4]. The difference in the values in our study and Garg RK et al[4] study remained insignificant statistically (P value >0.05).

Very low density lipoprotein (VLDL) level was 34.78 ±12.49 in haemorrhagic stroke patients in our study in comparison to 30.09 ±20.19 in the similar group in Garg RK et al[4] study while it was 32.59 ±13.01 in thrombotic stroke group in our study as compared to 32.71 ±18.92 in the same group of Garg RK et al[4] study. The difference between values of two studies was statistically insignificant (P value >0.05).

Conclusion:

We conclude that there is no differential effect of any lipid fraction in the causation of thrombotic and haemorrhagic stroke.

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