Afr. J. Med. med. Sci. (1990) **19**, 281–284.

Plasma lipids in Nigerian hypertensives

O. O. ADEDEJI* AND A. C. ONITIRI
Department of Clinical Pathology, Lagos University Teaching Hospital, PMB 12003, Lagos, Nigeria

Summary

In this study, the plasma lipids in 50 Nigerian hypertensives and 50 control subjects, matched for age and sex, were estimated by enzymatic colorimetric methods. Generally, the levels of plasma lipids for both controls and subjects were within the normal range. However, there were elevations of plasma total cholesterol (after correcting for the effect of weight), low density lipoproteins and triglycerides in the hypertensives. There was some decrease in the levels of high density lipoproteins (HDL) and cholesterol concentration in hypertensives but this was not statistically significant.

Résumé

Dans cette étude, les lipides plasmiques dans 50 cas d'hypertension des Nigérians et 50 sujets de contrôle correspondant en âge et en sexe, ont été évalué par les méthodes enzymatiques et colorimétriques. Généralement, les niveaux des lipides plasmiques pour les sujets de contrôle comme pour les hypertensifs étaient normaux. Néanmoins, il y avait des élévations de cholestérol plasmique total (après correction pour l'effet du poids), et les lipoprotéines et triglycérides d'une densité basse chez les hypertensifs. Il y avait une diminution dans le niveau de la concentration de lipoprotéine de haute densité (HDL) et de cholestérol dans les hypertensifs, mais ce n'était pas très significatif de point de vue statistique.

Introduction

Hypertension is now a common condition in African countries, especially amongst the urban populations [1]. Closely associated with hypertension is hyperlipidaemia. They are both risk factors in the development of atherosclerosis and its complications. However, very little is known about the incidence of hyperlipidaemia in the African communities. Onitiri *et al.* [2] showed that affluent Africans who consume diets rich in fats and proteins have significantly higher concentrations of serum lipids and lipoproteins than the low income group of Africans who consume diets rich in carbohydrates but low in fats and proteins. The same workers observed that contrary to the findings in Europeans the proportion of high density lipoproteins (HDL), on serum electrophoresis, was higher than that of low density lipoproteins (LDL). This observation supports the view that high serum HDL concentrations may be protective against ischaemic heart disease because its incidence is low in Africans. Contrarily, LDL has been implicated in atherogenesis.

The aim of the present study was to find the relationship, if any, between plasma lipids and hypertension. It was hoped that a better understanding of the association between these two factors might give information about the mechanisms of the atherogenic process as it begins to unfold specifically in the African. Furthermore, such information could contribute towards the diagnosis and management of both hypertension and hyperlipidaemia in Africans.

Subjects and methods

Subject selection

The subjects comprised 50 male and female patients attending the Hypertension Clinic at the Lagos University Teaching Hospital. Their mean age ± s.e.m. was 42.4 ± 1.7 years. An equal number of non-hypertensive male and female volunteers served as controls (mean age

*To whom correspondence should be addressed.

± s.e.m. 42.1 ± 1.7 years). Informed consent was obtained from all the participants. Both subjects and controls were matched for age and sex. The following parameters were measured in subjects and controls after an overnight fast: weight, height, blood pressure, plasma cholesterol, lipoproteins and triglyceride. Venous blood samples were obtained from the subjects.

The blood pressure was taken with the subject seated and with the aid of a standard aneroid sphygmomanometer calibrated against a mercury manometer. Boehringer-Mannheim colorimetric test kits were used, following the procedures established previously, to determine the concentrations of total cholesterol [3], triglyceride [4], LDL concentration by precipitating with calcium chloride/heparin, and LDL–cholesterol by complexing the cholesterol fraction with sulphuric acid/acetic anhydride [5]. HDL–cholesterol was also estimated using the Friedwald formula [6]:

$$\text{HDL–cholesterol} = \text{total cholesterol} - \text{triglyceride}/5 - \text{LDL cholesterol}.$$

A more accurate technique was used for the determination of HDL–cholesterol, i.e. the ultracentrifugation method [7].

For quality control, the accompanying Boehringer-Mannheim precinorm-U and precipath-U sera were used. Also, the blood glucose, uric acid, protein and sugar were checked routinely to eliminate those subjects who might have other problems apart from hypertension. Subjects chosen for this study had already been started on anti-hypertensive drugs. However, they were warned not to take their drugs for at least 12 h before these tests were carried out. Percentage weight was used as an index of weight:

$$\%\text{weight} = \frac{\text{weight in kg}}{\text{weight (cm)} - 100} \times 100.$$

All data were statistically analysed using correlation coefficient and Student's *t*-tests.

Results

The characteristics of the controls and subjects are shown in Table 1. There was no significant difference ($P > 0.05$) between their age and %weight. However, both diastolic and systolic blood pressures of the subjects were significantly different ($P < 0.001$) from those of the controls.

The plasma lipid concentrations of both the controls and the subjects are shown in Table 2.

Table 1. The characteristics of controls and subjects

	Controls	Subjects	*P*-values
%weight	105.8 ± 3.6	109.2 ± 3.1	> 0.05
Age (years)	42.1 ± 1.7	42.4 ± 1.7	> 0.05
Systolic pressure (mmHg)	115.0 ± 1.0	159 ± 2.0	< 0.001
Diastolic pressure (mmHg)	74.0 ± 4.0	105 ± 1.0	< 0.001

Values represent means ± s.e.m.

Table 2. Plasma lipids concentrations of controls and subjects

Plasma lipids	Controls	Subjects	*P*-values
Total cholesterol (mg/dl)	174.0 ± 6.0	181.0 ± 4.5	> 0.05
Triglyceride (md/dl)	77.9 ± 3.6	121.3 ± 5.0	< 0.001
LDL (mg/dl)	296.2 ± 14.0	382.2 ± 19.0	< 0.001
LDL–cholesterol (mg/dl)	102.1 ± 5.0	137.2 ± 6.3	< 0.001
HDL–cholesterol (mg/dl)	56.1 ± 5.2	48.8 ± 3.4	> 0.05

Values represent means ± s.e.m.

There were significant differences between the subjects and their controls for triglycerides, LDL and LDL–cholesterol ($P < 0.001$).

Discussion

This study has shown that the mean concentrations of plasma lipids, especially triglycerides and some lipoproteins, particularly LDLs and LDL–cholesterol, in hypertensive subjects are higher than those in non-hypertensive controls. The observation may not be surprising especially when one considers the role of plasma lipids in the development of atheromatous lesions which causes a reduction in arterial compliance and normally leads to an increase in the mean arterial pressure [8,9]. The mechanism involved in atheroma formation has been explained by Davignon [10] using the lipid hypothesis. It was postulated that accumulation of lipid involves at least three processes.

1. Transfer of lipids from blood to the arterial wall.
2. The binding and sequestration of lipids in the arterial wall.
3. The metabolism and removal of lipids from artery.

Total cholesterol

It has been found that the mean total plasma cholesterol concentration is not increased significantly in the hypertensives. Making allowance for the effect of anti-hypertensive drugs that the subjects were taking before the study, the increase might be much more considerable. However, cholesterol is known to play a central role in the development of atheroma in the arterial wall.

The evidence in support of this view includes the finding of cholesterol as a major constituent of atheromatous plaque; the observation in many epidemiological studies of a strong correlation between plasma cholesterol concentration and the prevalence of coronary atherosclerosis; and measures aimed at lowering plasma cholesterol concentration in experimental hypercholesterolaemia cause a reduction in the size of atheromatous lesions [10].

LDL and LDL–cholesterol

There was an elevation of the concentration of mean LDL and LDL–cholesterol in the hypertensive subjects. The cholesterol involved in atherogenesis has been shown to derive mainly from LDL [11].

Triglycerides

There was an elevation of triglyceride with an increase in blood pressure. The levels of increment in triglycerides amongst hypertensives compared to controls were more pronounced than the increment in cholesterol. This suggests that triglyceride measurement may be a better discriminant than cholesterol measurement between hypertensives and control healthy subjects. Although the role of triglycerides in atherogenesis is still not clear, it has, however, been postulated that lipoprotein remnants which are derived from triglyceride-rich lipoproteins might play a part in the development of atheromatous lesions [12].

HDL–cholesterol

There was a slight decrease in HDL–cholesterol of hypertensive subjects. The difference was not statistically significant. The mean values for HDL–cholesterol in both the subjects and the control group are within the protective limit (HDL–cholesterol value of 35 mg/dl [13]). These results support the view that the incidence of coronary atherosclerosis is low among Africans.

Conclusion

The basic testing programme when investigating disorders of lipid metabolism in arterial diseases should include determination of total cholesterol and triglycerides. In general, further diagnostic tests are not necessary when the total cholesterol and triglycerides values are below the normal range. HDL–cholesterol is useful in determining atherogenic risk. The combination of low HDL–cholesterol and high triglyceride levels is indicative of high risk of coronary heart disease. The ratio of total cholesterol to HDL–cholesterol has now been

shown to be an important diagnostic parameter for coronary heart disease, and also to be more effective than either of the variables alone [13].

References

1. Akinkugbe OO. High Blood Pressure in the African. Edinburgh: Churchill Livingstone, 1972:86.
2. Onitiri AC, Sander M, Boyo AE. Serum lipids and lipoproteins in healthy Africans. Clin Chim Acta 1977;81:57–61.
3. Siedel J. Enzymatic colorimetric method for cholesterol determination. J Clin Biochem 1978;19:838–40.
4. Weiland O. Glycerol. In: Bergmeyer HU, ed. Methods of Enzymatic Analysis. New York: Academic Press Inc. 1963:211–14.
5. Burstein M, Samaille J. Sur un dosage rapide du cholestérol lie alpha et aux beta lipoprotein res du serum. Clin Chim Acta 1960;5:609.
6. Friedwald WT, Levy RI, Fredrickson DS. Estimation of plasma low density lipoprotein cholesterol concentration without the use of preparative ultracentrifuge. Clin Chem 1972;18: 499–515.
7. Onitiri AC, Jover E. Comparative serum apolipoprotein studies in ischaemic heart disease and control subjects. Clin Chim Acta 1980;108:25–30.
8. Chajek-Shaul T, Eisenberg S, Hirsch A, Oliver-crona T. VLDL metabolism of phospholipids, cholesterol and apolipoprotein C in isolated perfused rat heart. Arteriosclerosis 1981;1:82.
9. Sodeman WA, Sodeman TM. Pathologic physiology. In: Mechanisms of Disease, 6th edn. Holt Sanders, 1979;198–229.
10. Davignon J. The lipid hypothesis. Pathophysiological basis. Arch Surg 1978;113: 28–34.
11. Goldstein JL, Brown MS. The low density liproprotein pathway and its relation to atherosclerosis. Ann Rev Biochem 1977; 46:897–930.
12. Zilversmit DB. Mechanisms of cholesterol accumulation in the arterial wall. Am J Cardiol 1975;35:559–66.
13. Assmann G, Schriewer J, Juchein KH. Zur Rolle des HDL-cholesterin in der Preventin medizin. Therapievoche 1981;31:5563–70.

(*Accepted* 8 *January* 1990)