SERUM LEVEL OF LOW-DENSITY LIPOPROTEIN CHOLESTEROL IN HYPERTENSIVE RETINOPATHY

B Badhu1, S Dulal1, N Baral2, M Lamsal2, JK Shrestha3 and S Koirala3

1Department of Ophthalmology and 2Department of Biochemistry, BP Koirala Institute of Health Sciences, Dharan, Sunsari; 3BP Koirala Lion’s Center for Ophthalmic Studies, Kathmandu, Nepal

Abstract. Increased serum level of low-density lipoprotein is associated with coronary artery disease. There are, however, no reports on whether the same is true in hypertensive retinopathy. A cross-sectional comparative study was carried out to evaluate the serum level status of low-density lipoprotein in hypertensive retinopathy, including 30 randomly selected subjects with hypertensive retinopathy; age and gender matched 26 hypertensives without fundus changes. Serum low-density lipoprotein cholesterol (LDL-C) levels were assessed in all subjects. Results showed statistically significant (p<0.0196) higher serum levels of LDL-C in hypertensive patients with retinopathy (mean ± SD = 2.45 ± 1.76 mmol/l, SE = 0.33 and 95% CI = 1.79 - 3.11 vs mean ± SD = 1.6 ± 0.4 mmol/l, SE = 0.08 and 95% CI = 1.44 - 1.76). An increased serum level of LDL-C is associated with hypertensive retinopathy.

INTRODUCTION

Prolonged uncontrolled systemic hypertension may result in visual impairment or blindness. It is known that hypertension aggravates atherosclerosis and vice versa (Mc Sween, 1992). Similarly, prolonged uncontrolled systemic hypertension results in retinal arteriolar sclerosis (Christlieb, 1994). Systemic hypertension is related to both the processes of atherosclerosis and arteriolar sclerosis.

A high blood cholesterol concentration in the form of low-density lipoproteins is the most important causal factor in atherosclerosis affecting the arteries of larger caliber and the central retinal artery before its bifurcation, whereas the arteriolar sclerosis develops in the retinal arterioles (Mc Sween, 1992). It, therefore, leads to hypothesize that the co-existence of atherosclerosis with systemic hypertension affects the development of arteriolar sclerosis and consequent fundus changes of the eye.

In view of the above, the present study was carried out to compare the serum level of LDL-C amongst hypertensive subjects with or without retinopathy.

MATERIALS AND METHODS

A cross-sectional comparative study was carried out in the Department of Ophthalmology including 56 subjects with systemic hypertension. They were labeled as hypertensive when their blood pressure was higher than normal when measured at least twice during two separate occasions after the initial screening. Diastolic blood pressure below 90 mm Hg with systolic blood pressure less than 140 mm Hg was considered as normal. Exclusion criteria were the presence of diabetes mellitus, cataract or conditions impairing fundal clarity. A detailed systemic and ophthalmic examination was carried out, including slit lamp biomicroscopy, and fundus examination under mydriatics with indirect ophthalmoscope, +90 D lens, Goldmann three-mirror lens and direct ophthalmoscope to identify fundus changes related to systemic hypertension. There were
Fifty-six subjects with systemic hypertension were included for evaluation and laboratory investigation for LDL-C. Thirty had hypertensive retinopathy; the remaining 26 had no ophthalmic evidence of retinopathy (Table 1).

As shown in Table 2, ten subjects had Grade III retinopathy, whereas twelve others had various ocular complications of hypertension.

Serum LDL-C levels were found significantly higher (2.45 mmol/l ± 1.76 vs 1.6 mmol/l ± 0.4) among the hypertensive subjects with retinopathy than among those without it (p < 0.0196) (Table 3).

**RESULTS**

Fifty-six subjects with systemic hypertension were included for evaluation and laboratory investigation for LDL-C. Thirty had hypertensive retinopathy; the remaining 26 had no ophthalmic evidence of retinopathy (Table 1).

As shown in Table 2, ten subjects had Grade III retinopathy, whereas twelve others had various ocular complications of hypertension.

Serum LDL-C levels were found significantly higher (2.45 mmol/l ± 1.76 vs 1.6 mmol/l ± 0.4) among the hypertensive subjects with retinopathy than among those without it (p < 0.0196) (Table 3).

**DISCUSSION**

It is known that LDL-C blood level is increased in patients with coronary artery disease. There are, however, very few reports on whether the same is true in the case of hypertensive retinopathy.

Akinkugbe (1989) reported that the generally better lipid profile in African blacks compared with whites is associated with a low prevalence of hypertensive retinopathy. It has also been established experimentally in hypertensive rhesus monkeys with atherosclerosis that marked increases in retinal hard exudates
Table 3
Serum level of low density lipoprotein in hypertensive subjects.

<table>
<thead>
<tr>
<th>Hypertensive subjects</th>
<th>No. of subjects</th>
<th>Range (mmol/l)</th>
<th>Mean (mmol/l)</th>
<th>SD</th>
<th>SE</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>With retinopathy</td>
<td>30</td>
<td>1.99-5.33</td>
<td>2.45</td>
<td>1.76</td>
<td>0.33</td>
<td>1.79-3.11</td>
</tr>
<tr>
<td>Without retinopathy</td>
<td>26</td>
<td>1.2-2.0</td>
<td>1.6</td>
<td>0.4</td>
<td>0.08</td>
<td>1.44-1.76</td>
</tr>
</tbody>
</table>

$t$-test for independent samples: $p < 0.0196$.

(lipid deposits) were found if the serum lipoproteins were high (Hayreh, Personal communication). Kohner (1995) has stated that raised LDL-C serum levels can be expected in patients with Grade III (KWB classification) hypertensive retinopathy.

This study showed that, among the 30 subjects with hypertensive retinopathy, 10 had Grade III severity and 12 others had ocular complications of systemic hypertension. When the means of LDL-C serum levels of the 2 groups with or without retinopathy were compared, the group with retinopathy had a significantly higher level ($p<0.0196$). The findings of this study, in conjunction with LDL-C serum level estimation, can be a useful tool in evaluating patients with systemic hypertension for the presence of retinopathy.

ACKNOWLEDGEMENTS

The authors acknowledge Prof MP Upadhyay for his constructive suggestions and encouragement.

REFERENCES


