Glycosylated hemoglobin and lipoproteins in patients with premature hair grayness

To the Editor

I read with interest the recent article "Glycosylated hemoglobin and lipoproteins in patients with premature hair grayness".[1] Hereby, I would like to elaborate important points and certain pitfalls in the interpretation of the data from chemical pathological point of view.

The authors focussed on measured fasting plasma glucose (not sugar), glycated (not glycosylated) hemoglobin and serum lipid profile including triglycerides, total and high density lipoprotein (HDL) cholesterol (not lipoproteins). They tried to correlate their biochemical indices to the development of premature grayness of hair (PGH). While they were successful in achieving statistically significant FPG and glycosylated hemoglobin (HbA1c) values in subjects (not patients) with PGH, however, inspection of the real values (as reflected by the mean and ranges) when compared with the desired values implemented by international bodies, appear to be quite normal. All FPG values were <110 mg/dl in PGH group (mean ± SD, 83.25 ± 8.67 mg/dl) and controls (76.6 ± 1.96) and so they were much lower than the cut-off threshold for FPG that is recommended by the World Health Organization and the American Diabetes Association.[2,3] A positive or even causative relationship would be expected with impaired FPG values 111-125 mg/dl and diabetic values of >126 mg/dl.[3] Hence, none of the PGH subjects had impaired fasting glucose state or even diabetes mellitus despite the highly prevalent nature of these conditions. In addition, despite the limited specificity of HbA1c in reflecting minor impairment of glucose intolerance and its influence by many factors affecting glycemic state and erythrocytes' life span,[4,5] however, the values of HbA1c were also within the lower normal range in both PGH group (4.8 ± 0.46%) and control (4.34 ± 0.21%). Accordingly, FPG and HbA1c values in subjects with PGH appear to be of no pathophysiological importance.

Similar principles appear to be valid for serum lipid profile in this study. Although several factors may influence the level of lipid components including physiological factors, particularly dietary and genetic, however, the quoted values of lipids in PGH and control groups are within the desired values recommended by the American National Expert Panel on Detection, Evaluation and Treatment of high blood cholesterol.[6] All subjects with PGH had total cholesterol < 200 mg/dl (range 140-160 mg/dl). Serum HDL-cholesterol values which are better reflected as HDL-cholesterol:total cholesterol ratio (or atherogenic index) were also within the desired ratio of <5.0 (range 1.3-4.57). Hence it appears that both total and HDL cholesterol are within the optimal levels and these values are even better than the reported population derived reference ranges when an estimate of up to 30% of normal population may be expected to have ranges outside these desirable levels.[7,8]

These indicators of glycemic and lipoprotein states in subjects with PGH are, therefore, within the acceptable reference ranges of normal population and they do not point to an underlying pathophysiological process.

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Reply from the author

Many thanks for the comments made by Prof. Mulla Abed regarding our article, HbA1c and lipoproteins in patients with PGH. We completely agree that HbA1c and lipoproteins are within the acceptable references range of normal population both in patients with PGH and control. But when these figures are compared with each other in statistical way, we found that patients with PGH had high figures. Bearing in mind that these parameters were positively correlated with the severity of PGH, this conclusion could be applied to any condition when there is a healthy control even if the findings are within the normal range.

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